EX LIBRIS
ROBERT L. PHILLIPS, M.D.

The Charlotte Medical Library
Charlotte, N. C.

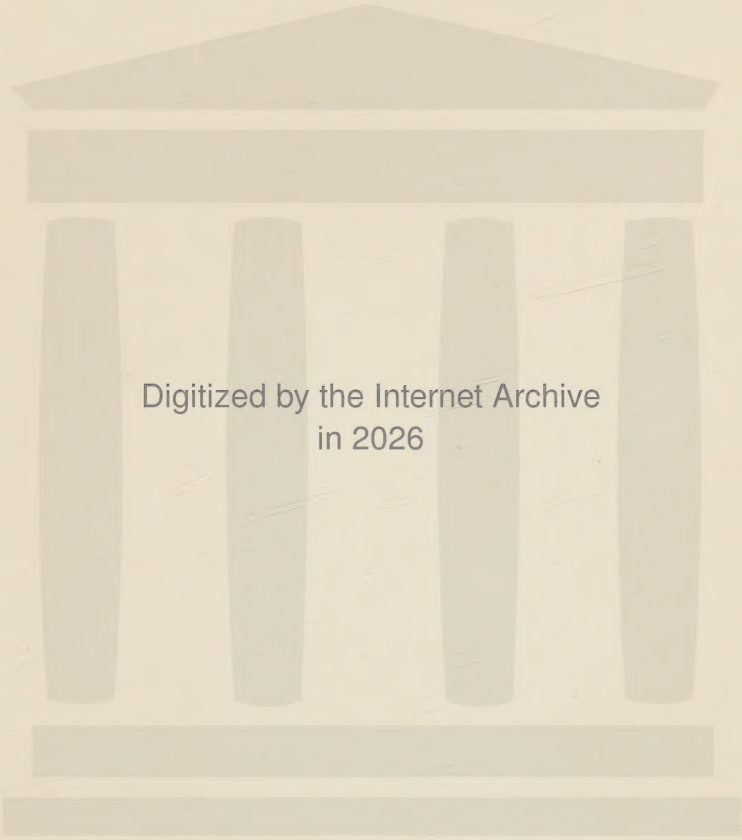
MEDICAL LIBRARY OF
MECKLENBURG COUNTY, INC.

The Charlotte Medical Library
Charlotte, N. C.

MEDICAL LIBRARY OF
MECKLENBURG COUNTY, INC.
DUPLICATE

MEDICAL LIBRARY OF
MECKLENBURG COUNTY, INC.

The Charlotte Medical Library
Charlotte, N. C.


Digitized by the Internet Archive
in 2026

https://archive.org/details/somefundamentalc0000vari_l1p9

SOME FUNDAMENTAL CONSIDERATIONS IN THE TREATMENT OF EMPYEMA THORACIS

SOME FUNDAMENTAL CONSIDERATIONS IN THE TREATMENT OF EMPYEMA THORACIS

BY

EVARTS A. GRAHAM, A.B., M.D.

MEMBER OF EMPYEMA COMMISSION, U. S. ARMY; PROFESSOR OF SURGERY, WASHINGTON UNIVERSITY SCHOOL OF MEDICINE; SURGEON-IN-CHIEF, BARNES HOSPITAL AND SAINT LOUIS CHILDREN'S HOSPITAL.

This essay was awarded the Samuel D. Gross Prize of the Philadelphia Academy of Surgery in 1920

ILLUSTRATED

ST. LOUIS

THE C. V. MOSBY COMPANY

1925

MEDICAL LIBRARY OF
MECKLENBURG COUNTY, INC.

Copyright, 1925, By The C. V. Mosby Company
(All rights reserved)

Printed in U. S. A.

Press of
The C. V. Mosby Company
St. Louis

FOREWORD

In presenting this little volume to the public the author is complying with the requirements of the trustees of the Samuel D. Gross Prize Fund. Its publication has been delayed until this time because of various disorders in the publishing business which existed at the time the prize was awarded. It was felt that if the work contained useful information for the medical profession it should be offered for sale at a reasonable price. Until now the publishers have felt that this could not be done.

The author wishes to emphasize that no attempt is made in the present work to cover the subject of empyema in an exhaustive manner. Even many of the details involved in the surgical treatment are given scant attention. As the title suggests, it is felt that the principles are the most important, and that if the treatment of empyema is conducted in accordance with the principles here discussed the mortality will be considerably less than that which existed before the recognition of the importance of these principles.

Since the work was prepared various criticisms of some of the theoretical aspects have been made. These are discussed in an addendum. It is hoped that, with the newer work presented here, the theoretical basis for the treatment of empyema will be established as sound.

E. A. G.

Saint Louis, February, 1925.

CONTENTS

INTRODUCTION

INTRODUCTION 13

SECTION I

PATHOLOGY 15

Importance of Avoiding Any Further Asphyxia, 18; Open Pneumothorax, 18; Normal Intrapleural Pressures, 19; Prevalent Conceptions of Mechanics of Open Pneumothorax are Erroneous, 20; Experimental Methods, 23; Equality of Relative Densities of Both Lungs in Unilateral Open Pneumothorax, 26; Experiments on the Living Dog, 27; Bilateral Pneumothorax, 34; Influence of Posture on Effects of Open Pneumothorax, 34; Relation of Amount of Air Entering Pleural Cavity to Amount of Air Entering Lungs, 35; Effects of Immobilization of Mediastinum and Adhesions, 41; Closed Pneumothorax, 43; Effects of Open Pneumothorax Other than Respiratory Disturbances, 48; Mechanism of Reaction to Change of Intrapleural Pressure not Clear, 49; Experimental Empyema, 50; Experimental Pneumonia, 54; Application of Experimental Results to Problem of Treatment of Empyema, 57; Clinical Confirmation of the Desirability of Avoiding an Open Pneumothorax in Acute Empyema, 59; Repeated Aspirations Compared with Early Continuous Drainage, 62; Deferred Operation, 63.

SECTION II

PREVENTION OF CHRONIC EMPYEMA 67

Objects of Treatment: To Sterilize and Obliterate Cavity, 67; Reasons for Failure of Cavities to Obliterate Themselves, 67; Desirability of Early Removal of Exudate from Surface of Lung, 69; Dakin's Solution Simultaneously Accomplishes Sterilization and Decortication, 69; Advantages of This Method over Former Methods of Treatment, 75; Common Operations for Cure of Empyema, 76; Disadvantages of Collapsing Operations, 77; Collapsing Operations Should Become Rare, 79.

SECTION III

CAREFUL ATTENTION TO THE NUTRITION OF THE PATIENT 81

SECTION IV

Discussion 84

Fistulas Which Communicate with the Lung, 84; When Is an Empyema Healed? 85; Adequate Drainage Desirable in Subacute and Chronic Stages, 86; No Discord between Experimental Results on Pneumothorax and Clinical Findings in War Wounds of the Thorax, 87.

SUMMARY AND CONCLUSIONS

Summary and Conclusions 91

ADDENDUM

Addendum 94

ILLUSTRATIONS

FIG.		PAGE
1.	Diagram showing the ordinary conceptions of the mechanics of open pneumothorax	21
2.	Tracing with left pleural cavity inflated	24
3.	Tracing with right pleural cavity inflated	24
4.	Tracing with inflated pleural cavities in recently killed dog	25
5.	Tracing made on living dog under ether anesthesia to show nature of reaction to open pneumothorax	30
6.	Tracing showing simultaneous changes in intrapleural pressure in dog	32
7.	Showing slightly advantageous effect of holding of mediastinum in case of large opening into left pleural cavity	43
8.	Adhesions between the left lung and the thoracic wall	44
9.	Closed pneumothorax in a dog under urethane anesthesia	47
10.	Photograph of membrane of old fibrous tissue removed from the lung in a case of empyema by the decorticating action of Dakin's solution	71
11.	Low power microphotograph of section of piece of tissue shown in Fig. 10	72
12.	High power microphotograph of a portion of the zone of inflammatory tissue	73
13.	Drawing to show the necrotic connective tissue next to the inflammatory zone	74

EMPYEMA THORACIS

INTRODUCTION

The epidemic of hemolytic streptococcus infections of the respiratory tract associated with empyema which ravaged the military camps of this country during the winter of 1917-18 did much to clarify the general understanding of not only the pathology but also the treatment of empyema. As a result of this opportunity of studying intensively and under proper control a very large number of cases, certain principles have come into general recognition which, although most of them are not new, have resulted in the establishment of more or less standard methods of treatment. In this respect the situation in regard to empyema has undergone much of the same transition as did the question of the treatment of appendicitis a few years ago in that now certain clearly established principles seem to warrant some degree of standardization of treatment. It is the purpose of the present article to dwell chiefly upon some of these principles rather than upon the details of the treatment. The great importance of them is shown by the fact that following their application in the army the mortality rate dropped from an appallingly high figure to one remarkably low. Before the war there was apparently but slight recognition of the fact that from the standpoint of mortality alone empyema constituted one of the most serious of the infectious conditions confronting the surgeon. In 1916 Lilienthal and Ware[1] recorded a mortality of twenty-one per cent in thirty-eight cases among children under twelve and a mortality of twenty per cent in twenty-five cases in adults. Wilensky[2] in a study of two hundred and ninety-nine cases at Mt. Sinai Hospital, N. Y., found a mortality of

twenty-eight per cent. More striking, however, than these figures was the remarkable mortality accredited to empyema in the military camps in the winter of 1917-18. The average mortality as noted in the replies of the various camps to the Surgeon-General's questionnaire of February 21, 1918, was 30.2 per cent, but in some of the camps it was well above seventy per cent. These high mortality figures occurred at a time when in most of the camps the former conventional method of early open drainage was in vogue. On the other hand, when this method was changed to the adoption of the cardinal principles to be discussed in this paper, the mortality dropped so low that at the hands of the Empyema Commission, for example, it reached the very low figure of 4.3 per cent,[3] and in fact it was the opinion of this commission that practically no cases of nontuberculous empyema uncomplicated by lung abscess, suppurative pericarditis, etc., would die if treated in accordance with these principles. The essential points of these new principles are: (1) Careful avoidance of open pneumothorax in the acute stage, (2) The prevention of a chronic empyema by the rapid sterilization and obliteration of the infected cavity, and (3) Careful attention to the nutrition of the patient.

SECTION I

PATHOLOGY

The rationale of the application of these principles is based largely upon the pathology present, and it is very important therefore to have in mind an accurate knowledge of the common pathologic findings in these cases. Prevalent statements in the literature, that a knowledge of the bacterial findings in empyema is unimportant, serve to show only that the authors of such statements are ignorant of the real importance of such information. As a matter of fact there is a very great difference both in pathology and clinical course between a pneumococcus and a streptococcus empyema and perhaps to some extent it was due to the ignorance of these striking differences that the mortality of the streptococcus cases was so appallingly high when conventional methods of treatment of pneumococcus empyema were applied to them. Pneumococcus empyema is almost always a complication of lobar pneumonia and it usually is not recognized until after the acute stage of the pneumonia has passed and at a time when probably the disappearance of much of the exudate from within the lung has increased the vital capacity* over what it was during the active acute stage of the infection. That this is a very important consideration will be shown later under the discussion of the effects of open pneumothorax. On the contrary in the hemolytic streptococcus infections, it is customary for a massive pleural effusion to occur early in the course of the disease, in fact so early that there has been a tendency among some to conclude that in certain cases the pleuritis with effusion antedates the involvement of the

*The term "vital capacity" used here and elsewhere in this article has the definite meaning to physiologists of the maximal amount of air which can be exhaled after a maximal inhalation.

lung. The pathology of this condition has been very admirably described by MacCallum,[4] and I shall quote extensively from his work: "Dyspnea of the most extreme type especially during inspiration is characteristic and results in a livid cyanosis. Many of the patients become hoarse or lose their voices entirely. They are alert, apprehensive, and sleepless, and there is often delirium." "Two main types of pulmonary lesions have been described, although these are frequently combined. The first, designated interstitial bronchopneumonia, is essentially the result of infection of the bronchioles with large numbers of hemolytic streptococci which leads to great changes in their walls and in the adjacent pulmonary tissue. The most striking accompaniment of this is a patchy atelectasis or collapse of the lung affecting those portions formerly supplied with air by the obstructed bronchi together with the formation of firm nodules of consolidation about the terminal portions of the bronchi. In addition to this there is extreme engorgement of the blood vessels of the lung and hemorrhage into its tissue about the nodules of consolidation. The walls of the bronchi themselves become densely infiltrated with mononuclear cells, and thickened. Extreme hyperemia and the new formation of blood vessels and of connective tissue add greatly to this thickening and to the deformity of the wall. In some cases the whole lining becomes necrotic and coagulated and constitutes a diphtheritic pseudomembrane. Corresponding with the changes in the bronchial wall are those in the walls of the adjacent alveoli which become greatly infiltrated with mononuclear cells, the alveolar capillaries are distended with blood, the epithelium is proliferated rapidly and desquamated into the alveolar cavity. These cavities are found to be filled in the neighborhood of the bronchus with blood, dense fibrin, a few mononuclear cells, and greater numbers of desquamated epithelial cells. Those which are more distant contain diminishing numbers of these cells but are filled with a thick viscid fluid. This

rather gelatinous edema gives a semblance of confluence between these areas of consolidation before it is really completed.'' ''Later the pleural exudate becomes more distinctly purulent, having begun as a thin greenish-brown turbid fluid filled with streptococci and disintegrated leucocytes and shreds of fibrin. Rapid organization of the inflammatory exudate is everywhere characteristic. This affects the alveolar and bronchial contents and they are replaced in part by connective tissue. It results particularly in interstitial new growth of connective tissue which thickens the alveolar walls, the bronchial walls, the adventitia of blood vessels, and the interlobular septa.'' ''Most striking of all is the rapid organization of the thick layer of fibrin which generally covers the pleura. This results in the formation of thick red vascular granulation tissue on the pleural surface. Adhesions between contiguous surfaces are quickly rendered permanent.'' ''Occasionally large portions of the inflamed lung become necrotic.'' ''The second type, designated arbitrarily lobular pneumonia, is composed of cases in which the streptococci invade in the same way. One does not know how to distinguish the symptoms and yet at autopsy, although sometimes even the lungs may look much the same at a casual glance, they are really different. There are no firm peribronchial nodules. Instead there are patches of consolidation in which the alveoli are filled with blood and leucocytes and enormous quantities of streptococci. * * * Widespread necrosis occurs with final destruction of areas of lung substance. Hemorrhage about these areas is usual and there is often pleural effusion but without any organization of the exudate. There are many cases in which this lobular pneumonia occurs in the pure form, but there are many others in which it is found side by side with the interstitial bronchopneumonia. They are different expressions of reaction to the same infection.''

Important points in the pathology to be emphasized from

the standpoint of treatment are the extent of involvement of pulmonary tissue in the streptococcus cases, the involvement of the bronchioles with sufficient exclusion of air to produce marked cyanosis and dyspnea, and the tendency to rapid organization of the pleural exudate. In contrast to the changes in the streptococcus cases are those found in the pneumococcus cases in which there is relatively less disturbance of the air-intake with less cyanosis and dyspnea and a much less rapid organization of the exudate.

Importance of Avoiding Any Further Asphyxia

These characteristic features of the pathology of the common varieties of empyema suggest at once that, particularly in the case of streptococcus empyema, it is of fundamental importance to avoid any additional element of difficulty of respiration in a patient already so dyspneic that he is cyanotic and suffering from air-hunger. An operation, therefore, which would establish an open pneumothorax in one of these acute cases would seem to be clearly contraindicated. To determine this point definitely, experimental work was carried out with the idea of first investigating the effects of open pneumothorax by using methods different from those which had been employed by previous investigators.[5] It was felt that prevalent conceptions of the effects of open pneumothorax were perhaps not entirely correct and that any additional light which could be thrown on the question would be of importance not only in connection with empyema but with thoracic surgery in general.

Open Pneumothorax

By open pneumothorax is meant a condition in which there is free entrance and exit of air into a pleural cavity through an opening in the chest wall. It is to be distinguished from a "closed" pneumothorax in which free air is enclosed in a pleural cavity with no exit to the outside

and from a "valvular" pneumothorax, a condition in which air may enter but not escape from a pleural cavity. The latter is a serious condition which is often found associated with pulmonary tuberculosis and is due to a direct communication between the lung and the pleural cavity. Throughout this article whenever the word pneumothorax is used without qualification, the condition of open pneumothorax is meant.

Normal Intrapleural Pressures

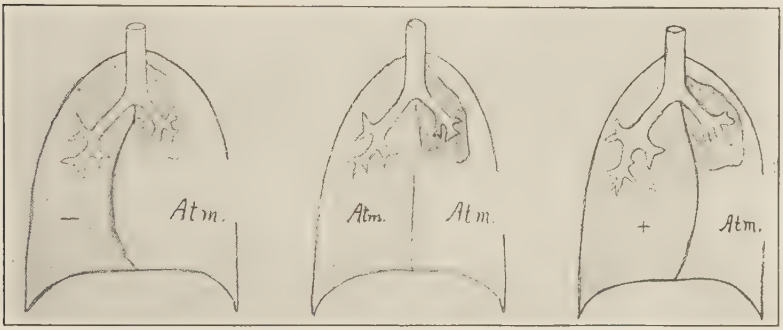
Under normal conditions the act of respiration depends chiefly upon an alternate expansion and contraction of the chest. The enlargement of the thorax during inspiration necessitates the inrush of air down the trachea into the lungs to equalize pressure within and without the lungs, and the corresponding contraction of the thorax results in an expulsion of air from the lungs. It follows, therefore, that during inspiration the pressure within the pleural cavities is less than that of the atmosphere, and it is commonly said that a "negative pressure" exists. This pressure has its lowest value at the end of inspiration. It begins to rise with the commencement of the expiratory act, but under normal conditions it does not reach that of the atmosphere even at the end of expiration, unless the glottis is closed. Various determinations of the intrapleural negative pressure have been made, but it is obvious that these are necessarily subject to rather wide variation. Donders[6] who was apparently the first to measure them in the human considered the normal intrapleural pressure at the end of expiration to be –7.5 mm. of mercury and at the end of inspiration to be –9 mm. of mercury. Aron,[7] in a living normal man, found an average pressure of –4.64 mm. mercury at the height of inspiration and –3.02 mm. at the end of expiration. More extensive reviews of these points may be found in the treatises of Emerson,[8] Sauerbruch,[9] and L. Mayer.[10] It is readily seen, therefore, that respiration can-

not go on unless at some time during the act of inspiration the intrapleural pressure becomes less than that of the atmosphere.

Prevalent Conceptions of Mechanics of Open Pneumothorax are Erroneous

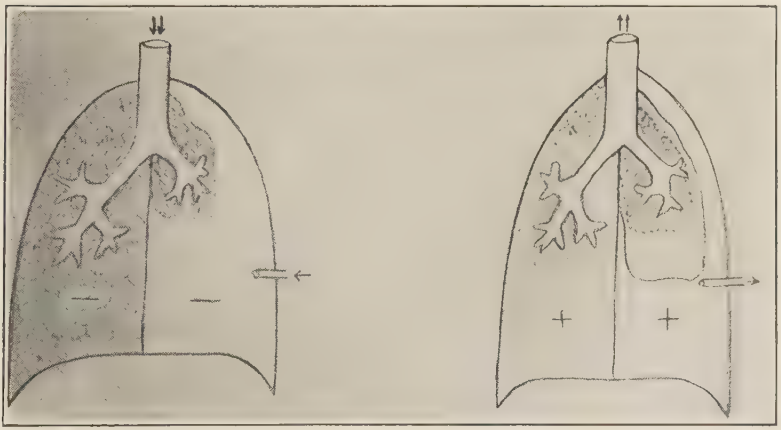
Largely because the idea has prevailed that any opening in the chest wall, no matter of what size, would result immediately in the establishment of atmospheric pressure within the pleural cavity, various conceptions of the effects of pneumothorax have arisen which are easily proved by experiment to be false. Indeed the prevalent conceptions are absolutely untenable when put to the test of direct experimentation. These conceptions are essentially that the mediastinum acts as a more or less rigid partition between the two pleural cavities and that when an opening is made into one pleural cavity the lung on that side becomes collapsed and respiration is maintained by the other lung. These ideas have become so deeply ingrained that all through the literature one finds references to the "collapsed" lung on one hand and the "sound" or "healthy" lung on the other. For example, Garrè[11] in a series of diagrams (see Fig. 1) to illustrate the conditions in open pneumothorax shows the mediastinum as a straight line, in cases in which the opening is small, with one lung collapsed and retracted and the other lung (on the unopened side) of normal size and apparently unaffected. In cases with a large opening, essentially the same condition is shown except that there is a slight bulging of the mediastinum away from and towards the opening in inspiration and expiration respectively. Likewise here also the lung on the side of the opening is shown as collapsed and contracted into a small mass about the hilum, whereas the opposite lung appears to be relatively unaffected. L. Mayer[12] summarizes from the literature the prevalent views on the changes produced by an open pneumothorax as follows:

"On the healthy side the modifications due to the pneumothorax with a small opening should adjust themselves by a deviation of the mediastinal pleura, the two surfaces of which are no longer submitted to conditions of identical



Inspiration | Normal expiration | Forced expiration

A. Respiratory mechanism in pneumothorax with large opening.



Inspiration | Active expiration

B. Respiratory mechanism in pneumothorax with small opening (Garré).

Fig. 1.—Diagram showing the ordinary conceptions of the mechanics of open pneumothorax. Taken from Garré.

equilibrium; on inspiration the lowering of intrapulmonary pressure of –7 mm. of mercury not manifesting itself on the fistula side, the mediastinal pleura should become curved

towards the healthy side and narrow the expansion of the other lung; the inverse should be produced at expiration. In reality these theoretical differences are minimal and are scarcely established by experimentation. If, on the contrary, the pleural opening is widely gaping the atmospheric air enters and goes out freely at each respiratory movement and the lung of this side will not be called upon at all to become distended. At each inspiration the pressure of the two sides of the mediastinal pleura will be different; on the healthy side negative pressure of 7 mm. of mercury, on the other side atmospheric pressure, with aspiration of the mediastinum towards the healthy side, towards which it ought to be markedly convex. In moderate and light expiration the pressure on the two sides remains equal; but if a forced or sudden expiration supervenes, the pressure is raised in the normal lung while it remains constant on the incised side, giving a convexity of the mediastinum on the side of the pneumothorax."

Direct experimental observations, which will be described in detail later, show that these conceptions are erroneous in that in the normal thorax the mediastinal structures offer so little resistance to changes of pressure that any slight or moderate change of pressure in one pleural cavity affects the other pleural cavity to practically the same extent. There is therefore practically an equilibrium of pressure throughout the normal chest and, from the standpoint of pressure relationships so far as moderate changes in pressure are concerned, the normal thorax can be considered as one cavity instead of two. When one pleural cavity is inflated with air at a known pressure of 10 cm. of water the pressure in the other pleural cavity is found to vary from 8 to 9.5 cm. of water. In other words, with so slight a pressure as 10 cm. of water the resistance of the mediastinum is equivalent to the pressure exerted by only 0.5 to 2 cm. of water. Reduced to terms of mercury this value is equivalent to only about 0.4 mm. or 1.6 mm. respectively and is there-

fore negligible. When subjected to such a pressure, therefore, one lung does not collapse to an appreciable extent more than the other, and the commonly supposed condition of collapse of one lung with maintenance of respiration by the other does not occur. Moreover, the pressure even in the opened pleural cavity does not come into equilibrium with that of the atmosphere until after death, owing to the constantly changing size of the thorax as a result of the respiratory movements. In this respect it is difficult for some writers to recognize the difference between a structure with fixed walls and one, like the thorax, which has moving walls. During expiration, the intrapleural pressure in open pneumothorax actually exceeds that of the atmosphere, but both theoretically and by actual determination the intrapleural pressure becomes less than atmospheric on inspiration.

Experimental Methods

To determine these points experiments were carried out in several different ways. Pressure changes induced by inflation of one pleural cavity with air were determined on human bodies immediately after death and then compared with similar changes observed in normal dogs immediately after they were killed with ether. When it was found that these agreed in all respects it was felt that conclusions drawn from experiments carried out on the living dog could be properly applied to the living human. In general the method which was used for the determination of these pressure changes was as follows:

A metal cannula (diameter 4 mm.) was inserted into each pleural cavity (usually in the fifth interspace in about the anterior axillary line). Each cannula was attached by rubber tubing of the same size and kind to a tambour, which, by means of an indicator, registered the pressure changes on a smoked drum. In one rubber tube was interposed a T tube, which was connected in one direction with

a water manometer and in the other with a rubber atomizer bulb. By means of the bulb, air was forced into the corresponding pleural cavity until the manometer registered

Fig. 2.—Tracing showing that when the left pleural cavity of a fresh adult human cadaver is inflated with air at a pressure of 10 cm. of water, the right pleural cavity registers a pressure of 9 cm. The top line is a record of a pressure of 10 cm. made with the tambour attached to the right pleural cavity. Calibration showed that the actual pressure was 9 cm. of water.

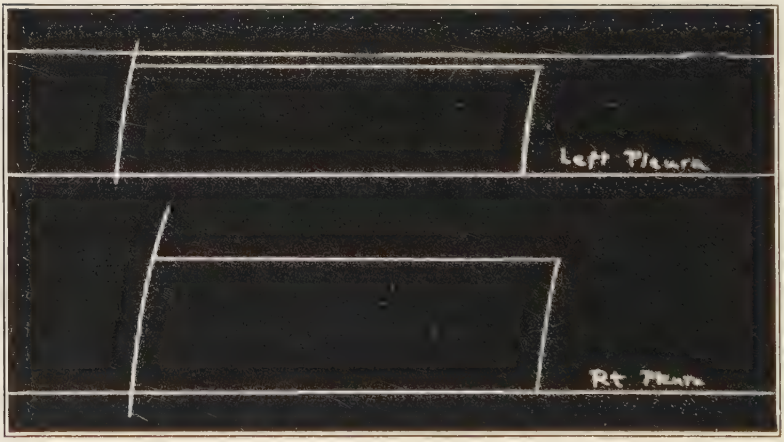


Fig. 3.—A similar tracing with the right pleural cavity inflated. Calibration showed that in this case also there was a difference in pressure of only 1 cm. of water (about 0.8 mm. of mercury).

10 cm. pressure. Simultaneously with the rise of the indicator on the tambour of that side the other indicator also became elevated above its base line. Calibration of the

reading of the second indicator showed the equivalent of a pressure of 8 to 9.5 cm. of water. In other words, when a known pressure, equivalent to 10 cm. of water, was established in one pleural cavity there was practically the same pressure in the opposite pleural cavity. This experiment has been carried out on five human cadavers, two of which were infants of four months and fourteen months respectively, one a child, aged fourteen years, and two adults. Duplicate determinations on the same cadaver sometimes showed a variation of 0.5 cm. in the reading of the opposite side, due probably to experimental error: that is, when

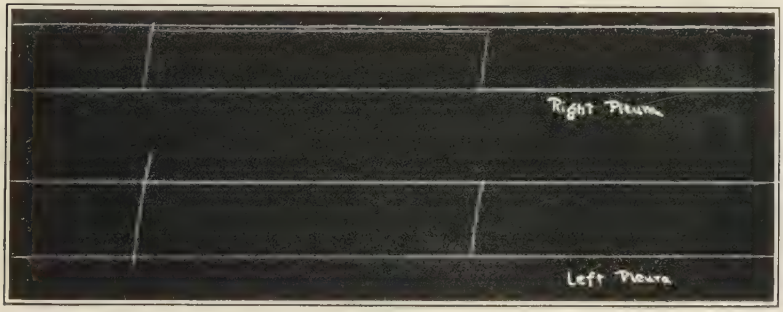


Fig. 4.—Tracing made in the same way with a recently killed dog, which shows that the dog is strictly comparable with the human, since here also the difference in pressure between the two pleural cavities amounted to only 1 cm. of water.

the pressure of the inflated side was the equivalent of 10 cm. of water, that of the opposite side registered sometimes only 9 cm. and at other times 9.5 cm. No difference was noted whether the right or left pleural cavity was inflated. These results were strictly comparable with those which were obtained on normal dogs which had been killed with ether. It is to be emphasized, however, that chilling of the dead body increases the resistance offered by the mediastinum. Cadavers which had been kept in the refrigerator for several hours showed a difference in pressure sometimes of as much as the equivalent of 3 cm. of water, but control dead dogs kept in the same refrigerator for the

same length of time always showed the same variations. Since the object sought in the experiments was really to determine the extent of the mobility of the mediastinum, it was necessary to rule out the possibility of the transference of pressure to the opposite side by being distributed through the diaphragm of the inflated side to the abdomen and from there through the diaphragm of the opposite side to that pleural cavity. In order to exclude such a possibility the abdomen was opened by a long median incision before inflation of the pleural cavity. By so doing the transfer of any extra pressure to the opposite pleural cavity by way of the abdomen was obviated. It is clear, therefore, that the distribution of the pressure from one pleural cavity to the other, under the conditions of the experiment, was due to the fact that the mediastinal structures were pushed over against the opposite lung.

Equality of Relative Densities of Both Lungs in Unilateral Open Pneumothorax

A more direct method of determining whether the lungs are equally compressed consists of measuring their relative densities when one pleural cavity is inflated with air at a known pressure. If the pressure relationships in both pleural cavities are affected about equally by altering the pressure in one, then both lungs should be about equally compressed and should show about equal relative densities. To determine this point experiments were carried out as follows: Normal dogs were killed with ether or chloroform and into one pleural cavity air was injected until the pressure was equal to that of 10 cm. of water. Through a low tracheotomy in the neck, plaster-of-Paris paste was then pushed down into each main bronchus in order to block its lumen and thus prevent the escape of any air from the lungs during the process of removing them from the chest. After allowing the plaster to "set" for a few minutes the lungs were then removed. In this way it was

possible to remove the lungs so that they remained in the actual condition in which they were before the thorax was opened. The weight and volume of each lung was then determined, the latter by measuring the amount of water displaced. The plaster was then thoroughly removed from the bronchi and its weight and volume subtracted from that of the corresponding lung. In this manner the specific gravity of each lung was approximately determined, which indicated fairly closely the amount of compression of each lung. When dogs are killed with ether or chloroform the condition of the lungs is not uniform even when nothing else is done to them. One lobe may be well inflated while another is partly collapsed even on the same side. The specific gravity of the two lungs may vary considerably. In some cases the left and in others the right lung is the denser. But when the lungs are collapsed by inflating one pleural cavity these variations are not so apparent. The experimental results on five dogs are as follows:

		Density of Lungs	
		left	right
No. 1.	Right pleural cavity inflated with air to a pressure equal to that of 10 cm. of water	.74	.74
No. 2.	Left pleural cavity inflated	.73	.69
No. 3.	Left pleural cavity inflated	.70	.72
No. 4.	Control, not inflated	.52	.48
No. 5.	Control, not inflated	.43	.51

By showing an equal amount of compression of both lungs when only one pleural cavity is inflated these results lend striking support to the conclusion drawn from the other experiments, viz., that, in the normal thorax, alterations of pressure in one pleural cavity are accompanied by practically the same changes in the other pleural cavity.

Experiments on the Living Dog

Experiments on the living dog not only confirmed the previous observations in dead dogs that when an open pneumothorax is made on one side there is a simultaneous

change of pressure to practically the same extent in the other pleural cavity, but they also afforded an opportunity to determine by exact observation the nature of the associated phenomena, such as the changed character of the respiration, the amount of dyspnea, the mechanism of the attempts at compensation, etc. It was felt furthermore that, since no differences were noted between the results obtained on the human cadaver and the dead dog, conclusions to be drawn from observations on the living dog could safely be applied to the living human. The method used was in general the same in all the experiments. It consisted essentially in recording simultaneously on a smoked drum the pressure changes in each pleural cavity and in the trachea and the changes in the amplitude and rate of the respiratory movements. To record the variations in pleural pressure the same cannulas as already described above were inserted into the pleural cavities and connected with Marey tambours by means of rubber tubing. The changes of tracheal pressure were recorded in practically the same way by attaching a glass T tracheal cannula to another tambour, and the variations in the rate and amplitude of the respiratory movements were noted by attaching a fourth tambour to a spring tambour, which was tied to the animal's chest. When an opening was made into a pleural cavity, therefore, its effects could be at once determined by the movements of the various indicators on the revolving smoked drum.

Most of the experiments were conducted under general anesthesia, either urethane or ether, but a few were performed with local cocaine anesthesia. No important difference was observed between the changes in pleural pressure produced by the open pneumothorax when an animal was under the influence of general anesthesia and those produced when the opening in the pleural cavity was made under merely local anesthesia with cocaine. The factor of general anesthesia, therefore, in most of the experiments can be

considered as having no appreciable influence on the results. In the control experiments with cocaine no attempt was made to record the tracheal pressure or the changes in the respiratory movements. In all, thirty-eight dogs have been used for the work on pneumothorax. In addition, similar experiments have been carried out on three cats and two rabbits. No essential differences were observed regardless of the kind of animal used.

A marked difference in the character of the reaction by the animal occurs, depending upon whether a small or a large opening is made in a pleural cavity. By small or large opening is meant small or large in comparison with the air-inlet to the lungs. Normally, the area of the air-inlet is the area of the open glottis, but generally in our experiments it was the area of the tracheal cannula. In the accompanying figure (Fig. 5) when the expressions "5 mm. opening" and "1 cm. opening" are made, diameters of approximately spherical openings rather than areas are meant, because at the time most of this work was done no planimeter was available for determining the areas of the openings. A more detailed discussion of the important relationship which exists between the amount of air which enters the lungs and that which enters the pleural cavity will be given later in this article. The characteristic effects produced by a pleural opening smaller than the air-inlet to the lungs may be summarized thus: Immediately upon making the pleural opening there is an increase of pressure in both pleural cavities. Whereas, before making the opening both the inspiratory and expiratory pressures are entirely negative, immediately afterward the pressure rises so much that it is almost entirely positive; the indicators descend below the base line of atmospheric pressure only at about the end of the inspiratory effort. During this time there is noticeable a slight pause early in the course of the expiratory movement just before the pleural pressure reaches that of an atmosphere in its ascent from the nega-

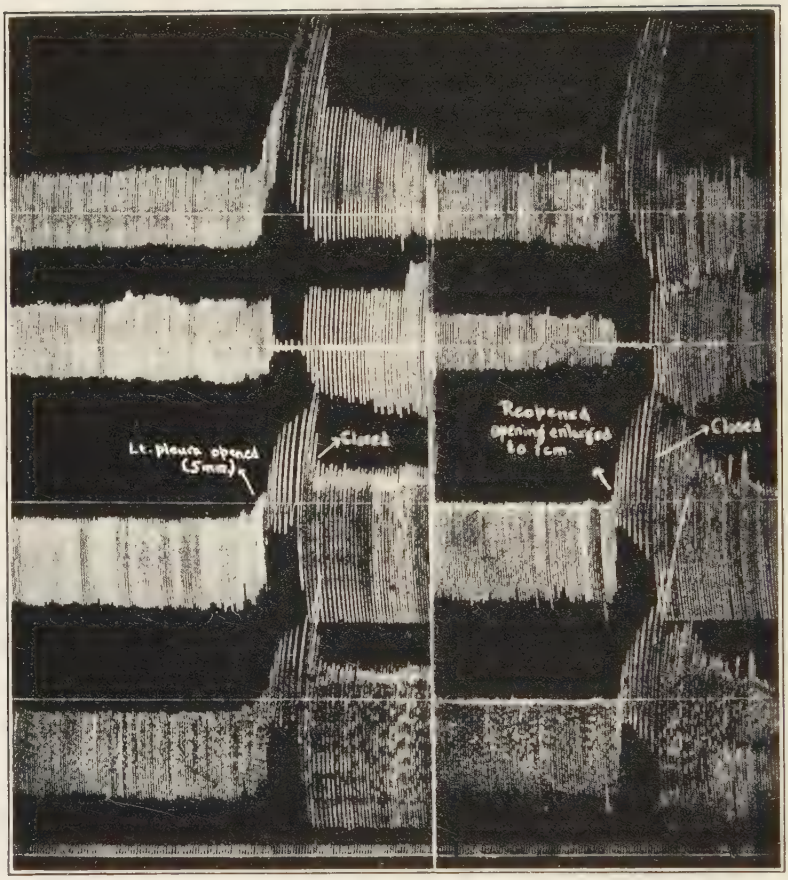


Fig. 5.—A tracing made on the living dog under ether anesthesia to show the nature of the reaction to an open pneumothorax with a moderate opening, as indicated in the changes in the respiratory movements, in the tracheal pressure and in the pressures in both pleural cavities. The upper tracing represents the respiratory movements, the next the tracheal pressure, the third the left pleural pressure and the fourth one, the right pleural pressure. The lowest line indicates the time in seconds. The base lines were drawn at atmospheric pressure. The break in the chart represents an interval of six minutes. Immediately after making the opening there is a simultaneous change of pressure in the pleural cavities from an entirely negative phase to one which is mostly positive. The respirations are slowed, but are increased in amplitude. In the unanesthetized animal, however, the respirations are accelerated as well as being increased in amplitude, after making the opening (see Fig. 6). Because the intrapleural pressure is largely positive, practically no air enters the trachea and the intratracheal pressure tends to be at equilibrium with atmospheric pressure. Immediately upon closure of the opening there is a simultaneous response in both pleural cavities, with restoration of negative pressure to a large extent, diminution of the amplitude of the respiratory movements and oscillations again of positive and negative intratracheal pressure, with inspiration and expiration. After an interval of six minutes, during which time probably most of the air in the pleural cavities has been absorbed, the intrapleural pressure has again become entirely negative.

tive phase. Simultaneously, also, the tracheal pressure changes in such a way that the indicator practically rests on the base line of atmospheric pressure instead of sweeping above and below the base line on expiration and inspiration respectively. This change is in accord with what is to be expected, since the tracheal pressure is a rough index of the amount of air passing down the trachea into the lungs, and since, obviously, with the pleural pressure almost entirely positive only a small amount of air can pass down the trachea into the lungs.

These changes are also coincidental with an effort on the part of the respiratory mechanism to compensate by making a greater effort to get air into the lungs; accordingly there is a marked change in the respiratory movement. As a rule, this change is rather an increase in amplitude of respiratory movement than an increase in rate. If the amplitude is increased considerably the rate will of necessity be slower than before. If the pleural opening is small enough to allow compensation, the pleural pressures will gradually diminish so that negative pressure will be reestablished and the tracheal pressure will rise simultaneously as the animal is enabled to get air into its lungs. The negative pressure, however, is never restored so completely as to reach the level at which it was before the opening was made, provided that the opening remains in free communication with one pleural cavity. Instead of both the inspiratory and expiratory pleural pressures being negative, as in the normal state, the pressure at the height of expiration will be positive. The ability of the animal, therefore, to withstand an open pneumothorax is dependent upon his ability to compensate by increasing his respiratory effort. Obviously a strong, vigorous animal can compensate better than a weaker one. In our experience young adult dogs have uniformly withstood the harmful effects of an open pneumothorax better than old or weak and emaciated dogs.

It is a very striking fact that usually the restoration of

Fig. 6.—Tracing showing the simultaneous changes in intrapleural pressure in a dog when an opening of 5 mm. was made in the left pleural cavity with cocaine anæsthesia. The base lines again represent atmospheric pressure. The upper tracing is the right pleural pressure and the lower that of the left pleural cavity. At *A* and *B* in each case the chest was opened and closed respectively.

negative intrapleural pressure can be instantly accomplished, even in cases in which because of the size of the opening there is no negative pressure, merely by closing the pleural opening, thus making a closed instead of an open pneumothorax, provided that the duration of absence of negative pressure has not been so great that because of asphyxia the animal is too nearly dead to compensate for the relatively slight disturbance resulting from the closed pneumothorax. But here, again, the intrapleural pressure does not become so low as it was before the opening was made, at least not within a few minutes. Presumably it would become normal as soon as the air contained in the pleural cavity was absorbed; but this point has not been tested. If the opening is made sufficiently large the animal will pass through a state of marked dyspnea and die of asphyxia within a few minutes, owing to his inability to compensate no matter how great his respiratory effort; for if the opening is so large that, despite his maximal respiratory efforts, he is unable to establish a negative pressure in the pleural cavity he will be unable to get air into his lungs, or if he is able to establish a slight negative pressure he may be able to get a little air into his lungs but not enough to maintain life. In this connection it is to be constantly remembered that an alteration of pressure in one pleural cavity is accompanied by an alteration of pressure to practically the same extent in the other pleural cavity, if there are no adhesions or no thickening of the mediastinal structures. No such condition can be recognized as the collapse of the lung on the affected side and maintenance of respiration with only the other lung, as is the prevalent conception of open pneumothorax. Instead, both lungs must functionate although both are compressed. In fact, if the opening into the pleural cavity be made in a favorable place for observation, and if it is not too large to prevent respiratory compensation, the lung on the affected side can be plainly seen to expand and contract with the inspira-

tory and expiratory efforts of the animal. Also, when a large opening is made, during the few minutes in which the animal remains alive the mediastinum can frequently be seen to flap from one side to the other with inspiratory and expiratory movements. It sometimes happens that, with a severe expiratory effort, the mediastinal pleurae are ruptured so that a double pneumothorax occurs. The various points about the changed pressure relationship may be best understood by reference to the accompanying reproduction of tracings.

Bilateral Pneumothorax

No more striking evidence of the incorrectness of the prevalent conceptions of open pneumothorax can be obtained than with experiments on bilateral pneumothorax. If it were true, as generally stated and believed, that an open pneumothorax results in the collapse of the lung on the affected side and maintenance of respiration with the other, then obviously a bilateral open pneumothorax should of necessity be fatal, regardless of the size of the openings. On the contrary, however, it is very easy to demonstrate that, provided the openings be not too large, a dog can tolerate a bilateral open pneumothorax indefinitely. Repeated experiments were performed to test this point and in every instance the result showed that there were no striking differences between the effects of a bilateral open pneumothorax and those of a unilateral open pneumothorax the area of the opening of which was approximately equal to the combined areas of the two openings of the bilateral pneumothorax.*

Influence of Posture on Effects of Open Pneumothorax

Elsberg,[13] as a result of experiments on dogs, has concluded that an open pneumothorax is tolerated better in a prone position, and he has approved the suggestion of

*If, however, the opening is a large one, then this no longer holds true. See in this respect the addendum.

Depage that in thoracic surgery the prone position should be the one of choice. Attempts to confirm these results failed in three experiments. Theoretically, it is very difficult to understand how an animal with an open pneumothorax could breathe more easily in the prone position. For in such a position it is necessary for the respiratory muscles to raise the trunk from the table with each inspiration. It seems clear, therefore, that, with this extra work to do, compensation for a pleural opening could not be made so easily nor maintained so long as in the supine position. Emerson[14] in his very comprehensive review of the literature of pneumothorax states with reference to the choice of position assumed, "while, as a rule, the patient prefers to lie on the affected side there are two remarkable cases on record in which the knee-elbow position was chosen." The individual presumably would choose whatever position would give him the most relief from his respiratory embarrassment, and the fact that there are only two cases on record in which preference was shown for a posture somewhat analogous to the prone would indicate that there is no decided relief gained usually from that posture. Probably the position which in a given case gives the greatest relief is somewhat dependent on the location of the opening and is brought about by the attempt on the part of the body to close the opening and thus to make a closed pneumothorax which, as will be shown later, causes the least respiratory embarrassment of all the types of pneumothorax if the amount of air enclosed is not too great. It seems possible that in the two exceptional cases quoted by Emerson the individuals had anterior openings.

Relation of Amount of Air Entering Pleural Cavity to Amount of Air Entering Lungs

The respiratory difficulties resulting from an open pneumothorax are dependent upon the relation of the amount of air entering the pleural cavity to the amount of

air entering the lungs. In the normal chest the action of the lungs, as a whole, proceeds practically as if no mediastinum were present, since the pressures in the two pleural cavities are always the same. If an opening is made into the chest cavity, part of the air which enters the chest during inspiration will pass in through the trachea and part will come through the chest opening. Only that part of the air which enters through the trachea is effective in carrying on respiration, and to prevent asphyxia the animal must increase either the rate or depth of respiration.

If we assume that the rate of respiration remains constant and that all compensation is effected by increasing the depth of respiration, then the change of chest volume which occurs at each inspiration must be made greater by the amount of air which enters through the opening. That is, if the animal requires a volume of air (T), at each inspiration, and if a volume (B) enters through the opening, then the increase in chest volume at each inspiration must be T + B after the opening is made. If, however, the rate of inspiration is increased after the opening is made the volume of air required at each inspiration will be reduced. If the rate was R_1 before and R_2 after the opening was made then a volume of only

$$\frac{R_1}{R_2} T$$

will be required at each inspiration, and the required increase in volume of the chest to secure the same amount of air per minute will be

$$\frac{R_1}{R_2} T + B$$

There is, however a limit to the possible compensation. The change in the chest volume from maximum expiration to maximum inspiration is the same as the "vital capacity" V.* The maximum possible compensation is reached when

*The vital capacity is measured on a spirometer. It is the greatest possible amount of air which can be expired after the greatest possible inspiration.

the increase in chest volume is V and when R_2 is the greatest possible rate comparable with this depth of respiration. Under these conditions it is evident that

$$B = V - \frac{R_1}{R_2}T$$

represents the greatest volume of air that can enter through the thoracic opening if the animal is to survive even for a short time. The anesthetized dog decreases the rate and increases the amplitude of his respiratory movements after the creation of an open pneumothorax. In the unanesthetized human, however, there is often an increased rate in open pneumothorax.

The ratio of the volume of air entering the chest through the thoracic opening to the volume entering the lung through the natural air passages would be equal to the ratios of their areas if it were not for two facts: (1) the natural air passages to the lungs have a considerable length which causes appreciable friction and a resistance to the entrance of air, and (2) the natural elasticity of the lungs offers further resistance. On account of these two factors an opening in the chest wall of a somewhat smaller size than the narrowest part of the natural air passages will admit the same amount of air. If the area of this narrowest part, which is the glottis, is C, and if the resistance to the passage of air makes this opening equivalent to an opening of an area aC located in the thoracic wall (a being less than 1), then the entire ratio of the volume of air entering the chest through the thoracic opening to the volume entering the lung through the natural passages will be

$$\frac{X}{aC}$$

X being the area of the thoracic opening. But from the equation above the maximum of this same ratio is

$$\frac{B}{\frac{R_1}{R_2}T} = \frac{V - \frac{R_1}{R_2}T}{\frac{R_1}{R_2}T}$$

and substituting, we have

$$\frac{X}{aC} = \frac{V - \frac{R_1}{R_2}T}{\frac{R_1}{R_2}T}, \text{ or } X = \frac{V - \frac{R_1}{R_2}T}{\frac{R_1}{R_2}T} aC$$

The maximum area (X) of an opening through the chest wall which can be withstood by an individual with a normal thorax (i.e., without adhesions or induration of the mediastinum) even for a short time can be approximately determined by substituting numerical values in this expression. The average vital capacity V and the tidal air T are given by Howell[15] as 3700 c.c. and 500 c.c. respectively. The normal rate of respiration R_1 during complete rest is about 15 per minute and the maximum rate, R_2, for the greatest possible depth of respiration is about 60 per minute.

$$\frac{R_1}{R_2}T$$

then equals 125 and

$$X = \frac{3700 - 125}{125} aC = 28.6\ aC$$

The area of the average human glottis when widely open may be taken as about 2.25 sq. cm. The value of a is assumed to be about 0.8 for the maximum rate and depth of respiration. This value must be less than 1 in order to allow for such factors as greater resistance to the passage of air down the trachea than through the thoracic opening because of greater length of the trachea, the presence of mucus on its walls, etc. Therefore aC = 1.8 and X = 51.5 sq. cm. In other words an opening of 5 × 10 cm. (2 × 4.1 inches) is

the largest for which compensation can theoretically be established in the average adult human even for short periods if the mediastinum has normal mobility.

It is to be emphasized that this value of 51.5 sq. cm. is the value of X in the average normal adult. Obviously, since the value of X depends to a considerable degree on the vital capacity, individuals with vital capacities greater than 3700 c.c. can withstand openings larger than 51.5 sq. cm. Peabody and Wentworth[16] have studied the vital capacity in a series of normal individuals. The tables on page 40 are quoted from their article.

For example, a man who, as quoted in the above tables, would have the unusual vital capacity of 7180 c.c., ought to be able to withstand an opening of 101.3 sq. cm., or 15.6 sq. inches. There is, therefore, no lack of agreement between the experimental results which are being recorded here and the findings in the war zone that openings in the chest wall which seemed surprisingly large were not necessarily fatal. It should be remembered also that what are apparently very large operative openings in the thoracic wall are often actually much smaller than they seem to be because of the presence in the incision of a lung which has been delivered out, gauze sponges, instruments, fingers of the operator, etc., all of which tend by their plugging to reduce the area of the opening and the amount of air which is sucked in at inspiration. Also, as will be shown later in experimental results, the partial fixation of the mediastinum exerted by the traction necessitated by delivery of the lung out of the incision is an aid in accomplishing compensation for the opening.

On the contrary, any condition which reduces the vital capacity or increases the demand for oxygen will of necessity diminish the size of the opening compatible with life. Such conditions are those which hinder the passage of air to the lungs (tracheal, or bronchial stenosis, etc.), those which reduce the available breathing space of the lungs

Normal Males

GROUP	NUMBER STUDIED	HEIGHT IN FEET AND INCHES	NORMAL VITAL CAPACITY	NO. WITHIN 10% OF NORMAL	HIGHEST VITAL CAPACITY	LOWEST VITAL CAPACITY	HIGHEST PER CENT	LOWEST PER CENT	NO. BELOW 90% OF NORMAL
I	14	6′ +	5100	9	7180	5030	141	99	0
II	44	5′ 8½″ to 6′	4800	41	5800	4300	121	90	0
III	38	5′ 3″ to 5′ 8½″	4000	31	5080	3450	127	86	1

Normal Females

GROUP	NUMBER STUDIED	HEIGHT IN FEET AND INCHES	NORMAL VITAL CAPACITY	NO. WITHIN 10% OF NORMAL	HIGHEST VITAL CAPACITY	LOWEST VITAL CAPACITY	HIGHEST PER CENT	LOWEST PER CENT	NO. BELOW 90% OF NORMAL
I	10	Over 5′ 6″	3275	5	4075	2800	124	86	2
II	13	5′ 4″ to 5′ 6″	3050	9	3425	2660	112	88	2
III	21	5′ 4″ or less	2825	16	3820	2500	135	89	1

(pneumonia, pleural effusions, etc.), those in which there is interference with the normal aeration of the blood or tissues (heart disease, acidosis, etc.), those in which there is interference with the normal action of the respiratory muscles (fatigue, prolonged surgical anesthesia, etc.), and those in which the level of metabolism is raised (toxemia, infection, etc.).

It should also be borne in mind that the value of X represents the approximate maximum opening compatible with life only so long as the respiratory muscles can maintain a maximum respiratory movement, and in addition, that it is only an approximation because of the variability in different individuals of some of the other factors, as for example, C.

In this connection it is probable that the frequent statements in the literature to the effect that conclusions on the results of open pneumothorax which have been based on animal experimentation cannot be applied to the human are due to the fact that the relatively small size of the laboratory animals has not been taken into consideration. For example, the vital capacity of rabbits and guinea-pigs must be so small that only relatively insignificant openings could be made in the chest wall without causing death. The situation in respect to dogs is only slightly better unless the animal is a large one. In the series of experiments here recorded it has not been possible to detect any fundamental difference between the phenomena of open pneumothorax in laboratory animals (dogs, cats and rabbits) and those in the human.

Effects of Immobilization of Mediastinum and Adhesions

Since the equalization of pressure in the thorax is dependent upon the mobility of the mediastinal structures, it would seem reasonable to suppose that any means of stabilizing the mediastinum would reduce the respiratory

difficulties by confining the effects of the pneumothorax to one pleural cavity. Old inflammation of the mediastinum with extensive induration will of itself result in a more rigid partition between the two pleural cavities. If, in addition to its being actually more rigid, the mediastinum is supported by strong adhesions, the effect of an open pneumothorax on one side will be still less felt on the other. This is the condition which is often encountered in performing secondary operations of the type of Estlander, Schede, Delorme, etc., on old cases of empyema, and its presence probably accounts for the fact that in these operations the thorax can be widely opened with much less danger of respiratory embarrassment than is the case with the relatively normal mediastinum. These same factors also tend to make the deferred operation in acute empyema more safe than one performed too early, particularly if the early operation is not done in such a way as to prevent an open pneumothorax. Furthermore, it can be shown experimentally that when the mediastinum is more or less fixed by pulling the lung well out of the pleural opening there is less pressure in the unopened pleural cavity than in the opened one (see Fig. 7). It is impossible, however, effectively to immobilize the normal mediastinum in that way because its extreme delicacy (especially in the anterior portion) allows it to be stretched and flapped about in spite of fixing it at one point by holding the root of the lung tight. It was hoped to demonstrate conclusively the influence which adhesions produced in various ways might have, but limitations of time prevented it. Fig. 8, however, shows the record of one experiment. A few adhesions were produced between the lung and thoracic wall by the injection of 1 c.c. of turpentine. The record shows that during the intervals in which the left pleural cavity was open the right pleural pressure was distinctly less affected. On inspiration, for example, the pressure was twice as low in the right pleural cavity as in the left. This obviously enabled the dog to

get more air into his lungs than he would have been able to do if the adhesions in the left pleural cavity had not to this extent prevented the aspiration of the mediastinum to the other side. Because of the elasticity of the lung tissue it is probable that extensive adhesions and those which involve the mediastinum itself are necessary in order to produce a more marked protective effect.

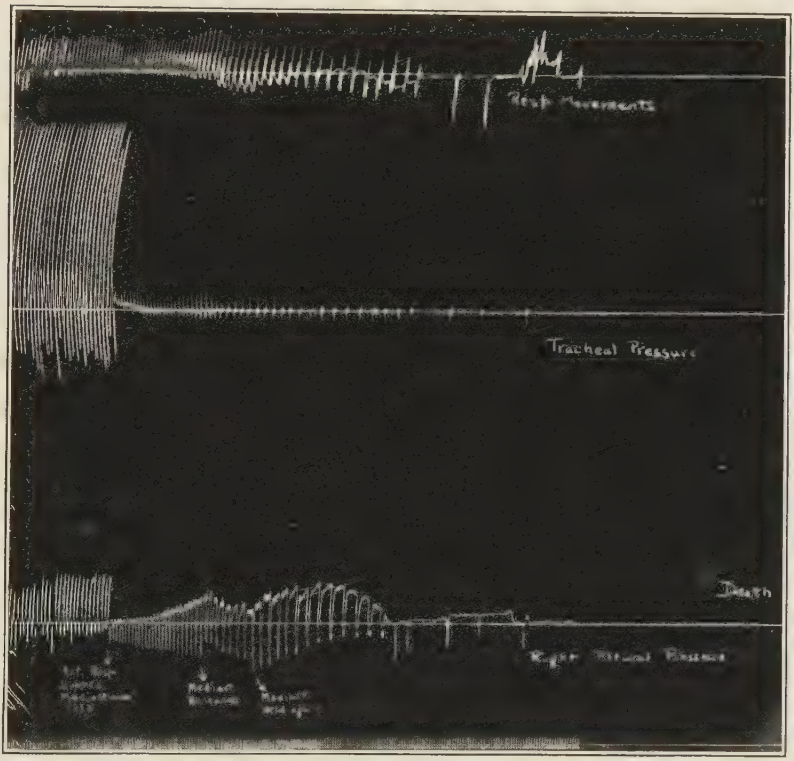


Fig. 7.—Showing slightly advantageous effect of holding of mediastinum in case of large opening into the left pleural cavity made by turning down a flap of pleura with six ribs while the dog was given artificial respiration by intratracheal insufflation.

Closed Pneumothorax

It has been a frequent and striking observation both experimentally and clinically that the dyspnea induced by

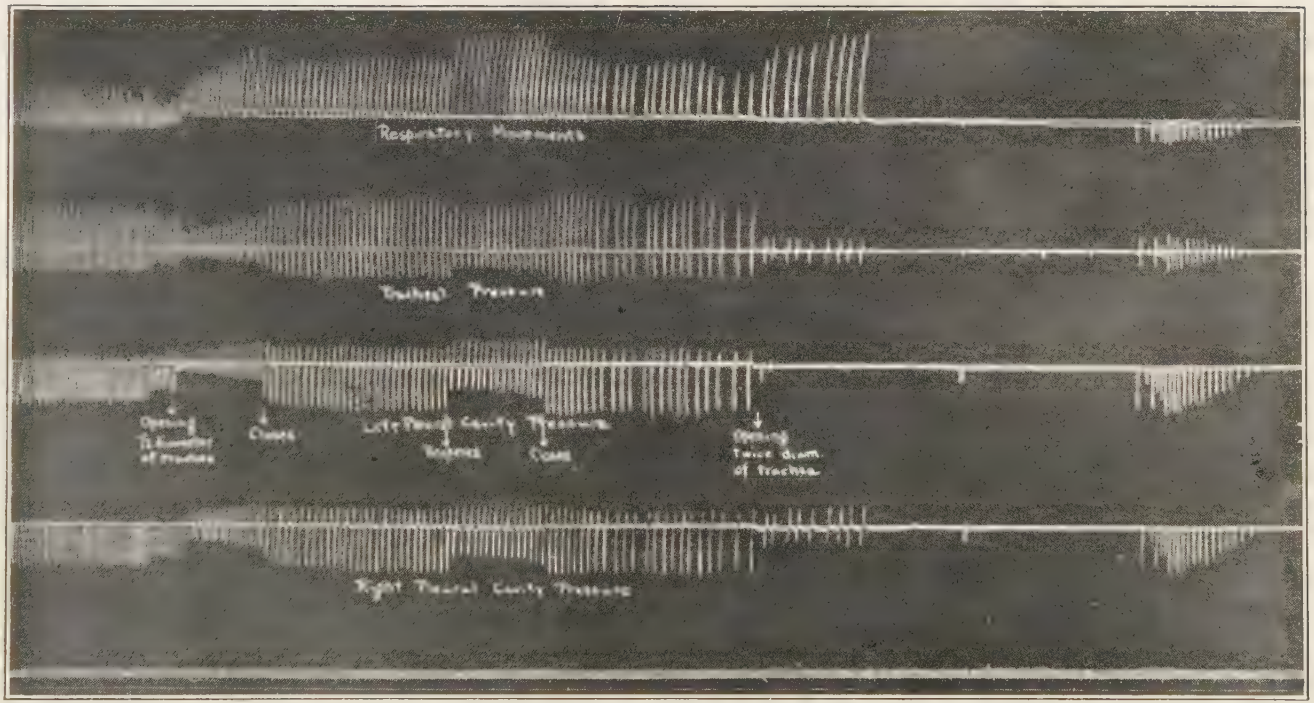


Fig. 8.—Adhesions between the left lung and the thoracic wall. Opening made into left pleural cavity. Dog under ether anesthesia. Because of partial fixation of the mediastinum by the adhesions the right pleural cavity is less affected than the left. Respiration is maintained longer with the right lung than with the left, and there is more negative pressure on the right side than on the left when the left pleural cavity is opened.

an open pneumothorax is relieved almost entirely by closing the opening. Since this procedure changes the conditions by merely making a closed pneumothorax instead of an open one, it is apparent that the closure of the wound is the important factor. Air is still retained in the pleural cavity, and, although it is absorbed by the pleura, it disappears only slowly, and the sudden benefit noted by the closure of the opening cannot be attributed to the immediate disappearance of the air. Apparently the explanation of the relative harmlessness of a closed, in comparison with an open pneumothorax lies rather in the fact that the ability to compensate for interference with the aeration of the lungs is limited. In a closed pneumothorax, no matter how much air is contained in a pleural cavity, no additional air can enter. It is necessary, therefore, only for the animal to increase his respiratory effort sufficiently to create enough negative pressure to allow him to take in the requisite amount of air to maintain aeration of his blood. Under conditions of rest this amount is equivalent only to the "tidal air," which in the human is about 500 c.c., or a little less than one-seventh of the average adult vital capacity. In those individuals whose vital capacities are greater than the average, obviously this fraction becomes still smaller, and in the exceptional man it may even be only one-fourteenth, since in general the value of the tidal air does not vary greatly with the value of the vital capacity. Accordingly, therefore, asphyxia should not occur until so much of the vital capacity has been replaced by the air in the pleural cavity that despite increased respiratory efforts the animal is no longer able to inspire the requisite amount of tidal air. When an open pneumothorax is converted into a closed one, particularly if the closure is made at the end of expiration, the amount of air enclosed in the pleural cavity is very much less than the amount required to prevent the inspiration of the necessary amount of tidal air; and naturally, therefore, there is very little dyspnea. On the other

hand in the case of an open pneumothorax we may think of an active competition for air going on between the glottis and the pleural opening. At each inspiration air not only enters the glottis but also enters the pleural cavity, and if the diameter of the pleural opening is the same as that of the glottis approximately the same amount of air will enter the pleural cavity as enters the lungs and the animal will be compelled to increase his inspiratory effort to get the tidal air into his lungs. If the opening is considerably larger than the glottis it will be still more difficult to get the required amount of air into the lungs, until when the pleural opening is of such size that with each inspiration enough air enters the pleural cavity to prevent the acquisition of the tidal air death will occur from asphyxia. Observations, both clinical and experimental, tend to confirm the truth of this explanation. For example, it is well known that an adult human can have as much as 2000 c.c. of fluid in his chest without producing alarming dyspnea so long as he remains at rest. Theoretically, an individual with an average vital capacity should be able to contain from 2500 to 3000 c.c. before developing a dangerous asphyxia if he remained quiet, and if it were not for the fact that his associated condition of toxemia and fever, uncompensated heart lesion, etc., demands a greater amount of oxygen than normal. Likewise, those with larger vital capacities should, for the same reasons, be able to contain pleural effusions of more than 2500 or 3000 c.c. depending considerably upon the amount of their vital capacities. Experimentally, I have injected into the pleural cavity of a dog of 8 kilos, through a small needle, as much as 1800 c.c. of air over an interval of twenty minutes without producing any marked asphyxia. Beyond that point, however, additional injections of only 50 c.c. at a time each had a very noticeable effect in increasing the dyspnea, and the animal died after about 2100 c.c. had been injected. The accompanying tracing (Fig. 9) illustrates the relative harmlessness of a closed

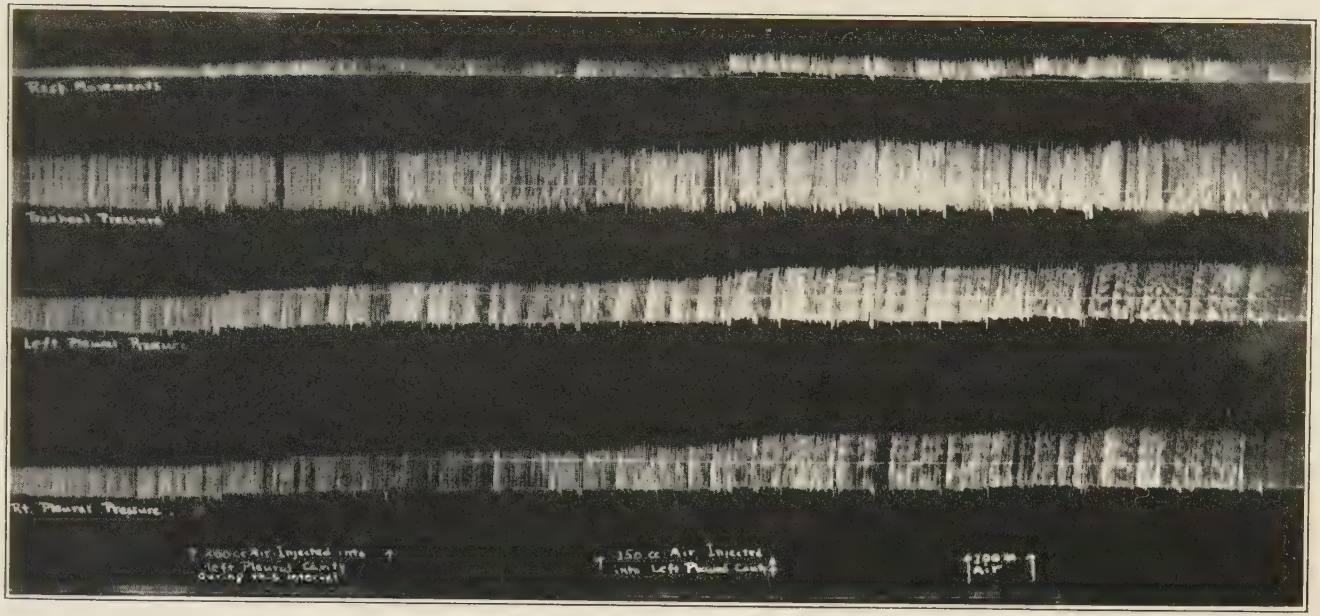


Fig. 9.—Closed pneumothorax in a 7-kilo dog under urethane anesthesia. It is to be noted that 450 c.c. of air can be injected into a pleural cavity without producing much distress, but again that the changes which result are strictly comparable in both pleural cavities.

pneumothorax in which a moderate amount of air is present. Lest it should seem surprising that a dog of 8 kilos could withstand so large an amount of air as 1800 c.c. in his pleural cavity, it should be recalled that in proportion to the size of the rest of the body the dog has a much larger thorax than the human.

Effects of Open Pneumothorax Other Than Respiratory Disturbances

In addition to the respiratory disturbances there are other marked effects of an open pneumothorax. Sauerbruch[17] has summarized them as heat loss, danger of infection and disturbances of the circulation. Important observations on the heat loss have been made by Sauerbruch. In unanesthetized rabbits in which he had established an open pneumothorax at a room temperature of from 20° to 22° C., the body temperature sometimes dropped as much as 3.5° C. within forty-five minutes. In dogs, also unanesthetized, within a half-hour he observed the body temperature fall 2° C. after making a pleural opening and rise again 1.6° C. within an hour after closing the opening. He also made the very striking observation that the heat loss in open pneumothorax is greater than in extensive laparotomy with eventration of the intestine for the same length of time. For example, a dog's temperature fell only 1° C. after having his intestine lying out of his abdomen for forty-five minutes, and a rabbit subjected to the same experiment showed a heat loss of only 1.3° C. after forty-five minutes.

The case of a secondary infection of the pleura in a condition of open pneumothorax is a fact already well known to those who have had much experience with empyema or with "sucking" wounds of the chest. This well known occurrence of infection subsequent to an open wound of the chest was held responsible for many of the deaths in the war, and putrefactive discharges are the rule in those cases

of empyema which have been treated by open drainage unless measures have been taken to prevent the secondary infection, as by the use of Dakin's solution, for example. It is easy to understand how the repeated aspiration of a considerable volume of air at intervals of many times a minute over a prolonged period of time would almost of necessity carry innumerable bacteria into the pleural cavity.

Sauerbruch has noted marked disturbances in the general circulation following an open pneumothorax. These are probably ascribable to the changed pressure relationships which interfere with the normal return of the venous blood to the heart. He states that "in pneumothorax the aspiration of the heart fails; a stasis results in the venous system. Measurements of the venous pressure give in fact an increase of the pressure." As a rule there is no noteworthy change in the arterial pressure. When asphyxia occurs there is evident a rise in the carotid pressure, as might be expected. Sackur[18] states that he has found a marked diminution in the amount of oxygen in the blood, in some instances to only one-half the normal amount.

Mechanism of Reaction to Change of Intrapleural Pressure Not Clear

The almost instantaneous response in the character of the respiratory movements which follows the creation of an open pneumothorax cannot be satisfactorily explained at the present time. It is very unlikely that asphyxia can account for it because the reaction occurs before any appreciable effect of asphyxia on the respiratory center can manifest itself. Also, that it is not due to the stimulation of nerve endings by the admission of cold air into the pleural cavity is shown by the fact that on several days when experiments were performed the temperature of the room was between 95° and 100° F. No noticeable difference was detected at this temperature and at lower temperatures.

Experimental Empyema

The general idea of the dangers of creating an open pneumothorax in the treatment of acute empyema was put to an experimental test in dogs in which an acute experimental empyema had been produced by injections of a virulent hemolytic streptococcus. It is evident that to be strictly comparable with the spontaneous streptococcus empyema of the human, the experimental empyema in the dog ought to be associated with the same pathologic conditions as the human. As was noted early in this article the human condition was characterized by the simultaneous presence of extensive pneumonia and a pleuritic effusion, a combination which necessarily would reduce greatly the vital capacity and would also, on theoretical grounds, make an open pneumothorax more dangerous to life than would be the case if the pneumonia were not present. But some difficulties were experienced in reproducing the typical pneumonia and empyema in dogs with sufficient regularity to make it possible to test the danger of open pneumothorax in empyema under conditions which were strictly comparable to the spontaneous cases occurring in man. Under the conditions of the experiment the advantage lay with the dogs rather than with the human, since because of the absence of any marked pneumonia in the majority of the experimental animals they should have been much more able to withstand an open pneumothorax than would be the case in the spontaneous condition occurring in the human. Despite these facts, however, it was clearly shown that the early establishment of open drainage is accompanied by worse results than if early drainage is not instituted. In a later part experiments will be detailed which show that the typical condition of pneumonia with its characteristic pleuritic involvement can be reproduced in some dogs in every way identical with the picture so often seen in man.

After several preliminary failures to produce an empyema by the intrapleural injection of from 10 to 15 c.c. of pure

broth cultures of a virulent strain of hemolytic streptococci which was furnished by Major R. A. Kinsella, a successful result was finally accomplished by injecting into the pleural cavity of a 6-kilo dog 30 c.c. of a broth culture of the same strain which had been made highly virulent for mice. This strain of streptococcus was one which had been isolated at autopsy from a man at Camp Lee who had not only an empyema but also suppurative pericarditis and multiple small lung abscesses. The dog which received this injection died about twelve hours later, and at autopsy the left pleural cavity (the injected side) was found to contain about 200 c.c. of slightly blood-stained, serofibrinous fluid which contained myriads of streptococci and a few necrotic leucocytes. This exudate resembled in every respect that which was obtained from the early human cases of streptococcus empyema at Camp Lee. Subsequently this exudate was used successfully in doses of from 1 to 5 c.c. for the production of empyema in a series of twenty dogs. In ten of the dogs intercostal pleural drainage with cocaine (0.5 per cent) anesthesia was established from four to twenty-four hours after the injection. To the other ten dogs which served as controls nothing was done. The operations were all carried out under strictly aseptic precautions and sterile dressings were later applied. A stiff rubber drainage tube with a one-quarter inch (6 mm.) lumen was used for each dog and the tube was stitched to the skin in order to hold it in place. The point selected for the drainage in each case was the sixth interspace in the anterior axillary line, since this seemed to be the most dependent portion of the thorax with the dog in the natural position. Sixteen of the dogs each received 2 c.c. of the exudate, two each received 3 c.c. and two each received 1 c.c. All the injections were made into the left pleural cavity. The dogs were paired as carefully as possible according to weights, so that each dog operated upon had a control which was not only of approximately the same weight

but which also had been injected with the same amount of pleural exudate. In general, out of each pair, the dog which seemed the stronger and the better operative risk was selected for the operation in order to give the operation every possible chance, since many of the dogs were poorly nourished and were victims of mange. In spite of all this, however, of the ten operated dogs all but one died (a mortality of 90 per cent) and of the unoperated dogs only seven died (a mortality of 70 per cent). Of the series of unoperated dogs, also, one fatality was in a dog which on the fourth day, while apparently well, was badly injured in a fight with another dog and received extensive lacerations in the neck, the back and the hind legs. Death occurred two days later and at autopsy the pleural cavity contained no exudate and appeared normal. It seems probable that it would not have died if it had not been for the wounds received in the fight.

It was a striking fact that of every pair of dogs which died the one which had been drained died from one to two days before its control with two exceptions, in which cases both the operated and the control dogs died at about the same time. It never happened that the unoperated dog of a pair died before the operated one. It is also noteworthy that the only dog of the operated series which survived pulled off its dressings and removed the tube on the next day after the operation. When seen shortly afterward (within three hours after the dressings had been pulled off) the pleural opening had already closed so that the dog no longer had an open pneumothorax. This was the only dog which disturbed its dressings. As a rule, death occurred in the operated dogs from forty-eight to seventy-two hours after the operation, and in the control series from the fourth to the fifth day. Of one pair, each of which had been injected with 2 c.c. of the pleural exudate, both the control and the operated dog died after about twenty-four hours following the injection. The operated dog in this case had

been drained four hours after receiving the injection. The operated dogs, as a rule, not only died more quickly than their controls but they all seemed much more sick than the unoperated dogs from the time that the pleural opening was made. They would lie quietly curled up, refusing food and resenting disturbance. The unoperated dogs were much more active. Immediately after operation each dog was placed in a cage by himself, was furnished with an abundance of water and food, and the cage was kept clean. In almost every instance the dressings were changed daily, dry sterile dressings being substituted for the soiled ones, with rigid aseptic precautions, even to the extent of wearing sterile gowns and sterile rubber gloves during the procedure. Each of the operated dogs had a profuse drainage of a thin serofibrinous discharge, sometimes slightly blood-stained, which microscopically showed innumerable streptococci and only a few leucocytes which were usually necrotic.

Each dog that died was examined at autopsy within a few hours after death. All of the operated dogs had been drained well, as shown by the absence of appreciable amounts of exudate in the pleural cavity. In practically every instance there was an extensive deposit of fibrin on the pleura, and there were many recent adhesions. In general there were more adhesions in the dogs that had been drained than in the controls. Two dogs showed in addition to a left pleuritis an involvement of the pericardium and the other pleura also. It was interesting that the pericardial fluid contained a noticeably smaller number of organisms than the left pleural fluid and that the right pleural exudate contained even fewer. In other words, it appeared as if the infection had passed right through the pericardium and mediastinum from the inoculated left side. A similar conclusion has been drawn by Stone[19] from observations on human autopsies. Of these two dogs, one had been drained and the other had not been. Three of the dogs that had been operated upon showed at autopsy pockets of pus be-

hind the upper part of the sternum which resembled very much those which had been described frequently in the human cases. This condition was not observed in any of the dogs that had not been drained.

The fact that in this series the dogs that had been subjected to early open drainage not only showed a higher mortality but that also from the time of the establishment of the drainage they were more sick than their controls to which nothing but the inoculation had been done, seems to establish, as well as any experimental results can, that early open drainage in empyema, at least in that type associated with hemolytic streptococci, not only accomplishes no good but is actually harmful. The experimental test therefore confirms the general theoretical deductions in the preceding portions of this article and coincides with clinical experience not only at our own hands but also at those of many others. Presumably the harm is produced by the open pneumothorax, with the train of resulting effects which have been discussed earlier in this article. Furthermore, the fact should not be overlooked that in this series of experimental empyema in dogs we were dealing almost solely with a pleuritic condition alone rather than with a combination of extensive pneumonia and pleuritic involvement. This is important to keep in mind because, as noted in the opening paragraph of this section, the threshold of safety for an open pneumothorax was greater in the case of these dogs than in that of human beings suffering from combined pneumonia and pleuritis. It was hoped to test out in another series of dogs the effect of early aspiration followed by late operation and of some of the various methods of continuous closed drainage but time was not available for the experiment.

Experimental Pneumonia

It has already been stated in the previous part that difficulty was experienced in regularly reproducing in dogs a

condition strictly comparable to the combination of pneumonia and empyema seen in the human. However, such a condition was produced in three out of six dogs. It was hoped to pursue this phase of the problem further in order to be able to test out the question of early open drainage on experimental animals which would be more nearly comparable to the human patients, but limitations of time have prevented it.

The method was intratracheal insufflation of pleural exudate from other dogs into a dog deeply anesthetized with ether. The amount of pleural exudate used has varied from 2.5 to 5 cm. One of the dogs in which a successful result was obtained also was moderately ill with distemper at the time the insufflation was made. A brief summary of one of the experiments with a gross description of the viscera of the dog as noted at autopsy is as follows:

An 8.5-kilo dog was given 2 c.c. of pleural exudate by the method just described above. Three days later the dog died. There was a very extensive pneumonia of both lower lobes with scattered areas of consolidation in the other lobes. Bilateral empyema (200 c.c. thin, slightly purulent fluid in left pleural cavity and 100 c.c. in right pleural cavity) was found, as well as suppurative pericarditis (about 50 c.c. of exudate in the pericardial sac similar to the pleural exudate) with extensive deposition of fibrin on the pericardium. Extensive fibrinous adhesions were present in both pleural cavities with massive fibrinous deposits on the posterior surfaces of both lower lobes. Each lower lobe also showed posteriorly a necrotic patch about 1 cm. in diameter which was found to allow air to escape when attempts were made to inflate the lungs with an atomizer bulb as MacCallum had done in his study of the pathology of the human lungs. It seemed in fact that on the right side there was a pneumothorax in addition to the empyema, as air apparently escaped from that side when it was opened. The tracheo-bronchial glands were much enlarged

and there was a moderate tracheitis. The other organs showed no important changes. The lungs were not cut but the thoracic viscera were placed *in toto* in Kaiserling solution. The lungs from two of the dogs were later studied by Dr. W. G. MacCallum who kindly furnished the following description of them:

"Incision into the several lobes on each side reveals areas of reddish-gray consolidation most extensive in the lower lobes. There is on each side in the central portion of the area in the lower lobe a ragged cavity filled with fluid and necrotic material and extending to the pleura, which is torn so that the abscess cavity appears in both cases to have communicated with the pleura. Other smaller areas appear opaque and necrotic, but are not liquefied. Microscopically it is found that the pleura is thickened and covered with a fibrinous exudate. No broad lymphatics are traceable into the substance of the lung. The bronchi are filled with leucocytes, among which are very numerous streptococci. Their walls are not markedly altered, although the epithelium is frequently desquamated. No conspicuous or thrombosed lymphatics are seen in their walls nor in the walls of the blood vessels. The alveoli are filled with an exudate of polymorphonuclear leucocytes, with some red corpuscles and great numbers of streptococci. Their walls are not infiltrated or thickened. Great areas in the midst of such consolidated tissue have become necrotic en masse and are surrounded by a zone of closely packed leucocytes. These areas, rich in streptococci, are sometimes intact, but the larger ones show a complete disintegration of the structure of the necrotic lung and of the enclosed exudate, and the softened, partly liquefied débris is seen to have retracted from the living tissue, which remains as a densely infiltrated wall. The consolidation has little in common with the interstitial bronchopneumonia observed in the recent epidemic, but is almost precisely like the condition also observed then which was referred to as lobular pneumonia.

In that condition large numbers of streptococci invaded the lungs, causing a widespread intraalveolar exudate, with hemorrhage and subsequent extensive necrosis of the infected tissue. The sections from the second dog repeat this picture so precisely that they need not be described."

Application of Experimental Results to Problem of Treatment of Empyema

As a result of the experiments previously described the dangers of an open pneumothorax seem now to be clearly established. Indeed it would seem apparent that an operation for empyema which carried with it the dangers of an open pneumothorax, if performed too early in the course of the disease, is accompanied by such very grave risks that the danger of harm from the operation outweighs any advantages which it may have. This is particularly the case in regard to the empyema due to hemolytic streptococci which accompanies the types of pneumonia described in the section dealing with the pathology of the condition. But presumably the same conclusions would apply to any type of empyema, especially if it is accompanied by a marked diminution in the vital capacity. If, however, the acute stage of the disease has passed and frank pus is present, there is comparatively little danger to be feared from an open operation for the purpose of securing free drainage, for the following reasons: (1) There is less danger of creating an open pneumothorax because, in our experience, there is likely to be a circumscribed abscess shut off by adhesions from any communication with the free pleural cavity so that during the operation the pleural cavity, properly speaking, is not entered. (2) Even if an open pneumothorax is created the patient is in much better condition to withstand its harmful effects, because (a) the subsidence of the active pneumonia has the effect of making the area of the air-inlet to the lungs larger than when many of the

MEDICAL LIBRARY OF
MECKLENBURG COUNTY, INC.

bronchioles and much of the lung parenchyma are blocked by the pneumonic process, so that the pleural opening is incapable of producing the same amount of harm, (b) the presence of adhesions and the inflammatory thickening and induration of the mediastinum tend to make it less mobile, (c) the patient's need of oxygen is less because of a more nearly normal metabolism, (d) the respiratory compensation is more efficient since, owing to a diminished toxemia, the respiratory muscles will not become so easily fatigued. (3) The patient is in better condition to withstand whatever shock there is connected with even so slight an operation as pleural drainage. (4) There is probably less risk of creating a septicemia from absorption of organisms from the fresh operation wound, an occurrence which seemed to me to have happened a few times at Camp Lee by the finding of positive blood cultures a few hours after operation in cases which previously had shown negative blood cultures, the operation having been done very early in the disease. As a matter of fact, a man who has recovered from his active pneumonic process and whose mediastinum has become thickened and stiffened by induration and exudate and immobilized by adhesions so that it no longer has normal mobility may withstand an open pneumothorax better than a normal man, for reasons shown in the experimental work.

If, in spite of a consideration of all the relative dangers of an early operation, it should be decided nevertheless to establish open drainage early in a particular case, it is obvious that only a small opening should be made during the stage of active pneumonia. Resection of ribs and the creation of large openings in the pleura should be reserved if necessary until later, after adhesions and induration have changed the mediastinum into a relatively immobile partition. In this connection it may be emphasized again that any obstruction of the natural air passages, any interference with maximum contraction or expansion of the chest

or any condition which increases the demand for oxygen will tend to decrease the limit of safety in the size of the opening. In the early stages of a streptococcus pneumonia several such factors are present. There is great obstruction of the air passages, and the vital capacity is reduced by the consolidation of the lung, the plugging of the branchioles, the exudate on the surface of the lung which limits its expansion and by the exhausted condition of the muscles of respiration. The amount of air required is greatly increased since the general metabolism is nearly doubled. All these factors tend to reduce the size of the thoracic opening for which compensation can be made by the patient. In fact, some cases have already so much interference with respiration that they are partly asphyxiated, and a thoracic opening of any appreciable size must inevitably cause death.

Clinical Confirmation of the Desirability of Avoiding an Open Pneumothorax in Acute Empyema

The desirability of avoiding an open pneumothorax in the removal of acute pleural effusions has long been recognized. It has resulted in the invention of many devices, such as the Potain aspirator, etc., which have as their object the accomplishment of drainage without the admission of air to the pleural cavity. In recent years, however, the principle of early open drainage in cases of acute empyema has come more and more into vogue. This tendency was due largely probably to the feeling that the treatment of acute inflammations of the pleura should be no different in principle from that established by long experience for acute inflammations elsewhere. It ignored the conditions which, as has been shown in this article both by experimental test on animals and by theoretical deductions, make of an open drainage in acute empyema a positively harmful procedure which must often be in itself an immediate cause of death. The very extensive epidemic of empyema in our military camps gave an opportunity for a clinical experiment on a

grand scale, and it has confirmed beyond any question of doubt the truth of the essential points which have been shown in the experiments recorded in this article. In the face of this overwhelming evidence it would seem that the operation of early open drainage of an acute empyema must be condemned as bad surgery.

At Camp Lee, Va., the Empyema Commission[20] established by the Surgeon General's office were able to reduce the mortality to 4.3 per cent from a very much higher previous figure (48 per cent) by instituting methods of treatment of which the essential point was the avoidance of an open pneumothorax. Similar striking reductions in the mortality occurred in other camps when early open drainage was abandoned. In a valuable statistical article Stone[21] has shown the marked reduction in mortality which occurred at Fort Riley, Kansas, after the early open operation was abandoned. His cases are divided into three series and the following quotation from his article will reveal the striking difference in mortality:

> "1. First series: Early operation (October 20, 1917, to January 21, 1918), 85 cases. Mortality, 61.2 per cent.
>
> 2. Second series: Early aspirations and late operation (January 12, 1918, to August 10, 1918), 96 cases. Mortality, 15.6 per cent.
>
> 3. Third series: Early aspirations and late operation (October 18, 1918, to February 14, 1919), 94 cases. Mortality, 9.5 per cent."

He also states that "the series are comparable as to type of infection." He ascribes the higher mortality of the second series over that of the third to the fact that probably the operation was postponed too long. Lest the reader should infer that perhaps the very high mortality in the cases of early operation was due to the unwise selection of

an extensive instead of a minor operation, the following table is quoted from Stone's article:

	Number	Deaths	Mortality Per Cent
Costectomy	50	28	56.0
Thoracotomy	32	21	65.6
Unrecorded	3	3	
	85	52	

Moreover, of this number of 85 cases, 71 were operated on with local anesthesia. It is evident, therefore, that the high mortality is not ascribable to the unwise selection of a too extensive operation nor to the unwise employment of general anesthesia. It seems more rational to ascribe it chiefly to the fact that an open operation was performed which carried with it the harmful effects of an open pneumothorax.

In the treatment of acute empyema an open pneumothorax may be avoided and yet drainage may be secured either by some method of continuous drainage by a closed suction apparatus or by repeated aspirations. Theoretically, it would seem as if early thorough drainage would be not only desirable but almost necessary. Yet its importance is apparently not so great as would seem to be the case, judging from the results obtained not only in our own cases but also in those at other camps when operation was delayed. Two cases at Camp Lee were seen each of whom had carried over 500 c.c. of pus in his chest for presumably several months. One of these cases was a negro who gave a history of a probable pneumonia in December, arrived at Camp Lee in April with a contingent of newly drafted men, and was able to drill for two days before being sent to the base hospital. At operation 600 c.c. of pus containing large numbers of hemolytic streptococci were evacuated, and the patient made an uneventful recovery. By June 10, he was entirely healed and had remained so six weeks later. It was the usual experience of the Empyema Commission to note a marked improvement in the general condition, even

sometimes with complete disappearance of fever, in patients who had had repeated aspirations but whose operations had been deferred until after the subsidence of the acute pneumonic signs, notwithstanding the fact that often at operation several hundred c.c. of pus would be evacuated. Stone[22] in his article also remarks upon the same finding in cases which had had repeated aspirations but whose operations had been deferred. Perhaps the explanation of the relative freedom from systemic effects of these retained accumulations of pus is to be found in the comparatively slight absorption of toxic material through the dense fibrinous exudate which surrounds and limits the abscess in these cases.

Repeated Aspirations Compared with Early Continuous Drainage

The relative advantages and disadvantages of the method of repeated aspirations as compared with the institution of an early continuous drainage with negative pressure will not be discussed in detail, since the object of the present article is the discussion of principles of treatment rather than details. There are certain considerations, however, which seem worthy of mention. Theoretically, the early establishment of continuous drainage with negative pressure should have the advantages of keeping the pleural cavity relatively free from liquid exudate and organisms, of aiding the expansion of the lungs as well as of avoiding an open pneumothorax with its attendant dangers. Practically, however, it seems doubtful if the results are appreciably better than with the method of repeated aspirations. There is also the additional risk, slight though it may be, that a delirious and unmanageable patient may by interference with the apparatus or the drainage tube allow air to enter his pleural cavity and may suffer from the effects of an open pneumothorax before it can be corrected. This danger was one of the considerations which deterred the Empyema

Commission from adopting as a routine the early establishment of continuous drainage with negative pressure. There is, moreover, the additional fact that experience has shown that a small proportion of these streptococcus cases will recover merely by aspiration. This number amounted to 13 per cent of the cases observed by the Empyema Commission, and Stone reports that 11.3 per cent of his cases recovered by aspiration alone and without operation. It is recognized also, however, that repeated aspirations are not without danger. It is quite possible that the point of the needle, if it penetrates the lung, may inoculate virulent organisms directly into the substance of the lung and induce abscess formation. If early continuous drainage is to be carried out it would seem that the simplest possible procedure should be employed, as for example, some one of the methods which are based on the principle of the insertion of a drainage tube through a cannula between the ribs with the later withdrawal of the cannula.*

Deferred Operation

If the method of repeated aspirations, followed by a deferred operation is used, pertinent questions of course are how to decide when to operate and what kind of an operation should be performed.

Practically, the decision as to when to operate is not difficult; and in all probability a difference of a few days on one side or the other is not of very great importance. A convenient criterion to take is the presence of frank pus; for almost certainly by the time the exudate has changed from its initial serofibrinous nature to a distinctly purulent quality the pneumonic involvement has practically cleared up, the vital capacity is probably increased, the patient's resistance is better, the abscess is walled off, and the general condition has improved as shown by a diminution of fever,

*Diederich in the winter of 1917 at Camp Pike was one of the first to use such a method in connection with the army epidemic.

absence of delirium and a decrease in the rates of the pulse and respirations. The Empyema Commission found that usually a period of from two to three weeks elapsed from the time of the onset of a streptococcal pneumonia to the appearance of definite frank pus, and on an average about four aspirations had been made. It is probable, in the light of the experimental work on pneumothorax, that a more accurate criterion of a safe time to operate would be furnished by actual determinations of the vital capacity in a given case. But no spirometer was available during the time that most of the clinical observations were made, and as yet there is an insufficient amount of data on hand to warrant our making any definite conclusions on this point. It is hoped that in the near future it will be possible to publish actual observations in this connection. Presumably, while the vital capacity remains very low, an operation with the establishment of open drainage would be dangerous.

In selecting the type of operation to be performed certain considerations are worthy of mention. In the first place it is to be emphasized that since the main purpose of an operation during the acute or subacute stage of an empyema should be to provide drainage, any measure which does not accomplish adequate drainage falls short of its object. Methods of treatment, therefore, which consist merely of the insertion of a tiny catheter to allow the installation of an antiseptic fluid into the pleural cavity at intervals but without offering an opportunity for the free escape of contained exudate are almost certainly bound to fail in the majority of instances. The most nearly ideal of any antiseptic solution known at the present time for such a purpose (Dakin's solution) loses its effectiveness very quickly when in contact with the albuminous contents of an exudate, and drainage away of the old solution with frequent installation of fresh unchanged antiseptic is very important. More will be said about this later in the next section. If an early operation is to be done, therefore, it would seem desirable

to choose a method which would permit continuous free drainage under negative pressure, combined preferably with the installation of Dakin's solution at frequent intervals. Many devices exist which permit continuous free drainage under negative pressure and the installation as often as wished of any desired quantity of an antiseptic fluid without interference with the air-tight connections of the apparatus. Since such a method predicates a relatively small opening in order to keep the connections air-tight, the type of operation preferably should be a simple intercostal opening into which the tube can fit snugly rather than a rib resection. Also the facts should not be forgotten that such an apparatus may be disturbed by a delirious patient with perhaps a resulting open pneumothorax, that there is a tendency for all tubes, however snugly applied at first, to work loose, and that, if rubber dam or some similar substance is used to aid in making the site of entrance of the tube into the chest wall air-tight, secretions tend to collect beneath the material and to result in excoriation of the skin.

If, on the other hand, the method of deferred operation is selected, consideration can more appropriately be given as to whether simple intercostal drainage or the resection of a rib should be performed. There is no doubt of course that under ordinary circumstances the creation of a larger opening, as by rib resection, will afford better drainage, but yet the conditions present in a case of empyema differ from those of an ordinary abscess of the soft tissues or even of the peritoneal cavity. As has been mentioned elsewhere in this article, the danger of establishing a general open pneumothorax as the result of the deferred operation is slight because by this time circumscribing adhesions have formed which restrict the abscess from the free pleural cavity; yet the possibility of this danger exists because it is conceivable that the adhesions already formed may break down. Moreover, the actual drainage of an empyema cav-

ity is facilitated by suction on the tube, and continuous suction is difficult to accomplish if the opening is too large because of the difficulty of securing air-tight connections for the tube in the wound. Another fact also is important in this connection, namely, that infection of the exposed cut ends of the ribs occurs with a resulting osteomyelitis of the rib which often persists for some time after the empyema, properly speaking, has healed. The possibility of an occurrence of an osteomyelitis of the rib can be minimized if sterile bone wax is rubbed into the open cut ends of the rib before the pleura is opened. In our experience, unless bone wax is packed into the open ends of the rib, this osteomyelitis is the rule after rib resections for empyema, and probably it occurs constantly. Still another point of considerable importance is that frequently small chips of bone drop into the pleural cavity either at the time of operation or later as the result of being separated off from the rib by the inflammation. These sequestra are troublesome, and they are responsible for some of the chronic sinuses in old empyema cases which would otherwise be well. They are difficult to detect and are therefore difficult to remove.

Such considerations as the above led the Empyema Commission in general to adopt a simple intercostal operation. If in a particular case better drainage seemed advisable later, the opening was enlarged by the resection of one or two ribs. Usually, however, the simple intercostal drainage was found sufficient.

In conclusion it may be said that, in spite of ardent debates between those who advise the routine performance of rib resection as a primary measure and those who with equal vehemence urge the primary employment of simple thoracotomy, the question is not a vital matter if in either case the operation is performed after the acute pneumonic involvement has subsided. But if an operation is performed early in the disease, it is undoubtedly wiser not to make a rib resection, for the many reasons discussed above.

SECTION II

PREVENTION OF CHRONIC EMPYEMA

Objects of Treatment: To Sterilize and Obliterate Cavity

In the successful treatment of any case of empyema there are two main objects: (1) the sterilization of the cavity, and (2) the obliteration of the cavity. Unless these two objects are accomplished permanent healing does not take place. If we wish to avoid failures or to protect ourselves against false hopes concerning a temporary apparent cure, it is essential that we keep these points constantly in mind. Up to this time we have confined ourselves in this article chiefly to a discussion of one of the important factors in the accomplishment of sterilization of the cavity, namely, drainage. Before the war almost the only means known of sterilizing empyema cavities was drainage aided by mechanical removal of thickened exudate by simple irrigation. Various antiseptic solutions had been tried but had been found, for various reasons, to be unsatisfactory or of no marked value. The relatively large number of cases which became chronic and which later had to be subjected to various types of mutilating operations bears evidence of the generally unsatisfactory results of these older methods. Many of these patients became chronic cases doubtless because neither of the two essential objects of the treatment had been accomplished. The cavity had been neither sterilized nor obliterated.

Reasons for Failure of Cavities to Obliterate Themselves

The failure of these cavities to obliterate themselves in all probability is due chiefly to an inability on the part of the lung of the affected side to expand sufficiently to fill up

its side of the thorax. This inability to expand is dependent chiefly on two factors, (1) fibrosis of the lung as a result of inflammation and (2) the thick, inelastic coat of exudate which covers the exposed surface of the lung and limits its inflation. This thick coat of exudate may be likened to a tightly fitting corset around the lung which obviously will interfere with its expansion, although usually the fibrosed pleura does not surround the lung but only affects a portion of its surface.

Evidence of the failure of the lung to expand properly is presented at autopsies on cases of unhealed empyema. It is revealed by finding that the lung on the affected side is smaller than the opposite one, and that the exposed surface of lung is coated with a dense layer of exudate which is sometimes fibrinous and sometimes organized connective tissue, in association frequently with a lake of pus contained within the thorax. The inequality in the size of the two lungs has frequently been ascribed to the collapse of the lung on the opened side from open pneumothorax, and it has even been used as an argument against the validity of the conclusion of the equilibrium of pressure throughout the thorax as developed in the experimental part of this article. It seems more probable, however, that there are other explanations. In the first place, as MacCallum has pointed out, "widespread necrosis occurs with final destruction of areas of lung substance." As this necrotic lung tissue is removed by expectoration and by gradual absorption, a considerable amount of fibrosis must occur during the healing process, which must necessarily result in the lung's being smaller than normal. At the same time the opposite lung may undergo hypertrophy, since the presence of long-standing adhesions and inflammatory induration have immobilized the mediastinum to such an extent that the healthy lung is no longer subjected to compression by slight changes of pressure in the affected pleural cavity. The remaining air-containing portions of the affected lung,

however, are not able to undergo hypertrophy to the same extent as they might otherwise do because of the thick coat of organized inelastic exudate which inhibits an adequate inflation of this lung.

Desirability of Early Removal of Exudate from Surface of Lung

It is obvious therefore that the early removal of this membrane is highly desirable. Because, at least in the streptococcus cases, organization of this exudate is known to occur early, it is clear that, other things being equal, the longer it is allowed to remain on the lung, the longer it will take to obliterate the cavity. For the cavity is obliterated only when the lung is in contact everywhere with the parietal pleura. In the past, recourse has been had chiefly to mechanical means for the accomplishment of this purpose, as, for example, operations of the type devised by Fowler and Delorme; but, although in some cases they have yielded brilliant results, on the whole they have been unsatisfactory. They have been associated with a high mortality and frequently they have not cured the patient. The fundamental defect in the rationale of such procedures is that attention is directed to only one of the two main objects of the treatment of empyema, namely, only to the obliteration of the cavity. It is fully realized of course that nature's efforts at sterilization are greatly aided by the obliteration of the cavity, but the frequency with which empyema recurs after the lung has apparently expanded to fill up the pleural cavity is evidence of the desirability of more active efforts directed towards the sterilization of the cavity.

Dakin's Solution Simultaneously Accomplishes Sterilization and Decortication

In neutral 0.5 solution of sodium hypochlorite (Dakin's solution), on the other hand, we have an agent which, on theoretical grounds, should be nearly ideal for accomplish-

ing the two main objects of the treatment of a case of empyema. For not only will it accomplish sterilization of the cavity but also, by its solvent action on the exudate, it will effect decortication and thereby obliterate the cavity. Clinical experience based on hundreds of cases in army hospitals not only at our own hands but also at those of other observers have seemed to indicate strongly that in the overwhelming majority of cases, if properly used, it will accomplish these purposes. The details of its use will not be discussed here. For these the reader may refer to the report[3] of the Empyema Commission or to the many articles on empyema which have been published during the last two years.

As a rule the removal of the restricting exudate from the lung is a gradual process which is apparently effected by its solution. In some cases, however, this membrane is removed in one large mass suddenly or in several smaller pieces. As evidence of the remarkable "decorticating" ability of the Dakin's solution an epitome of a striking case is here presented.

Pvt. G., 22 yrs. old, was wounded in the arm and leg in October, 1918, in the Argonne. He developed a pneumonia on the left side followed by empyema in November. He was drained through a thoractomy incision. When first seen by me on June 2, 1919, at U. S. A. General Hospital 28 at Fort Sheridan, Illinois, he had a small opening in the seventh interspace in the left posterior axillary line which was draining a considerable amount of pus containing large numbers of hemolytic streptococci. According to the patient's own account Dakin's solution had been used on him occasionally at some of the various hospitals in which he had been but never systematically. He was markedly underweight, had a septic appearance and had an afternoon temperature which ranged from 101° to 102°. He had entirely recovered from the wounds of his extremities. A measurement of his empyemic cavity showed that it had a

capacity of 350 c.c. and the patient stated that a measurement which had been made two months previously had shown the same capacity. Under local anesthesia a portion of the seventh rib was resected, and immediately after the operation instillations of 200 c.c. of Dakin's solution at two hour intervals were begun with provisions for adequate free drainage. On the sixth day a large piece of tissue was found plugging the drainage opening. A photograph of

Fig. 10.—Photograph of membrane of old fibrous tissue removed from the lung in a case of empyema by the decorticating action of Dakin's solution. For description see text.

this tissue after it had been hardened in formalin is reproduced here. (Fig. 10.) A description of it will be given later. Two days after the expulsion of this tissue the empyemic cavity would hold only 35 c.c. of fluid instead of 350 c.c. and no growth of bacteria was obtained from the secretion. Ten days later he was entirely healed and exam-

ination both by ordinary physical means and with the x-ray revealed no cavity remaining. On August 28th, he returned from a month's furlough, apparently entirely well and having gained 35 pounds in weight. Two months later I heard from him again, and he was still entirely well. The rapid decortication with later sudden expansion of the lung to fill

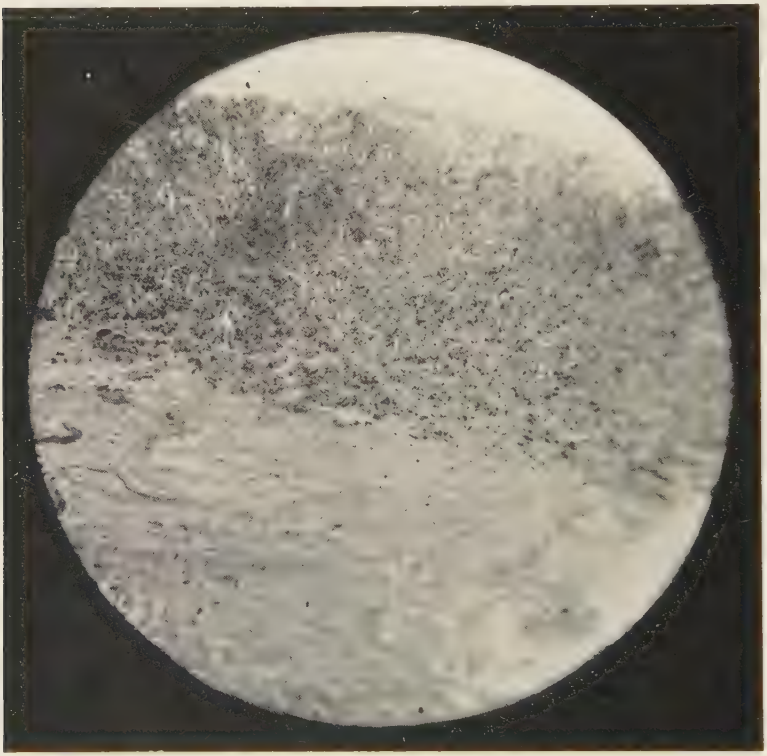


Fig. 11.—Low power microphotograph of section of piece of tissue shown in Fig. 10. Note the zone of inflammatory tissue next to the lung and the bands of connective tissue in the peripheral portion.

the cavity such as happened in this case is unusual, but yet I have observed it several times.

This piece of tissue was irregular in outline and flat. In longest diameter it measured nearly 6 cm. and in widest diameter about 3 cm. It was nearly 0.5 cm. in thickness.

It was yellowish-white, smooth except for folds in its surface, and in gross appearance it closely resembled fibrin. In fact it was considered to be fibrin until microscopic sections were made. The microscopic examination, however, showed that the main portion consists of necrotic organized tissue. Except for a narrow strip of inflammatory tissue which evidently indicates the side attached to the lung the whole

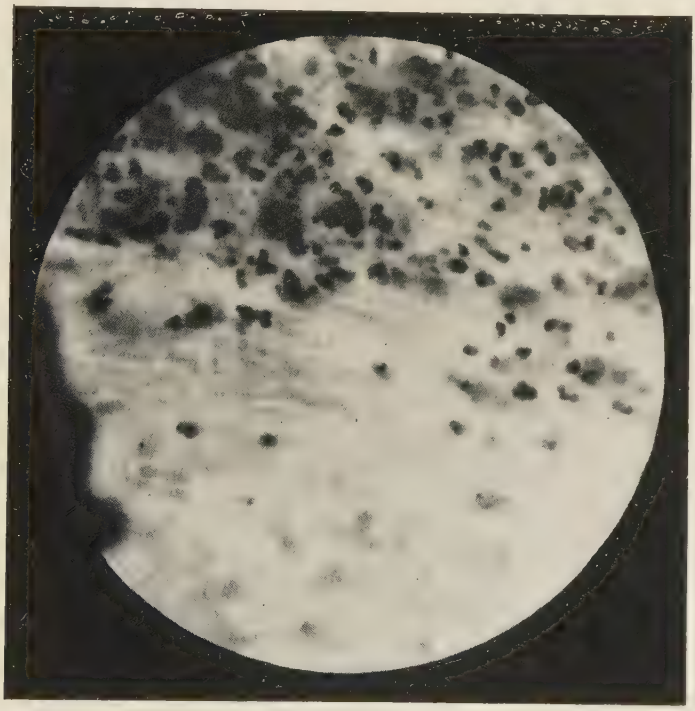


Fig. 12.—High power microphotograph of a portion of the zone of inflammatory tissue.

section is occupied by bands of wavy fibrils, arranged parallel to one another, with here and there the faint remaining outlines of what apparently have been blood vessels. The entire absence of nuclei except in the inflammatory zone is striking. These fibrils are readily stained by van Gieson's stain for connective tissue, but they are not

Fig. 13.—Drawing to show the necrotic connective tissue next to the inflammatory zone. This necrotic connective tissue was readily stained by the van Gieson method and was not stained by Weigert's stain for fibrin.

stained by Weigert's method for fibrin. Undoubtedly, therefore, the tissue is chiefly fibrous connective tissue which has become necrotic and is not unorganized fibrin. Throughout the inflammatory zone, and occasionally in the area just beyond, are seen irregular clumps of organisms. Many of these can be clearly distinguished as streptococci, and they are Gram-positive. The accompanying illustrations (Figs. 10, 11, 12 and 13) show the principal features of this tissue which have been discussed here.

The striking feature of this case is that the decortication of a considerable amount of organized exudate from the surface of the lung was accomplished quickly and easily with Dakin's solution, and that its removal was followed by an amazingly rapid expansion of the lung.

Other procedures of value in hastening the obliteration of the cavity are those measures which by physical means tend to increase the inflation of the lung. For example, blowing against resistance is of great value. This exercise can be carried out in a number of ways, blowing fluid from one bottle to another, inflating toy balloons, etc. Graded calisthenic, or "setting-up," exercises are likewise valuable in accomplishing the same purpose, although caution must be exercised in order not to make such exercises so violent as to overtax the heart, since nearly always some degree of myocarditis is present.

Advantages of This Method over Former Methods of Treatment

It goes without saying that any method of treatment which will accomplish the two principal objects, of sterilization and obliteration of the cavity, in the shortest time and in the greatest proportion of cases is the method of choice. We believe that a method based on the principles which have been emphasized above in this article will continue to show its superiority over methods which ignore these prin-

ciples, and we feel confident moreover that older commonly used methods of treatment of empyema will be more and more discarded. Based on the extensive experience afforded by the large number of cases of empyema in the army, it is felt furthermore that careful avoidance of open pneumothorax in the acute stage and the intelligent and systematic use of Dakin's solution will not only strikingly reduce the high mortality but will also make chronic cases relatively rare. Either of these accomplishments will more than justify the substitution of these principles for older existing methods. Stevens[23] has shown that of 56 cases healed under simple drainage without irrigations there were 14 recurrences, or 25 per cent; but of 67 cases healed under the Dakin-Carrel treatment with drainage there were only 8 recurrences, or 12 per cent.

Common Operations for Cure of Empyema

In this connection it is not out of place to call attention to certain facts concerning the common operations for the cure of chronic empyema. These operations are based chiefly on the idea of the desirability of obliterating the cavity, recognizing the well-known fact that it is all but impossible to maintain in a sterile condition a cavity surrounded by more or less rigid walls, particularly if it communicates with the outside air. In general two different methods of operative procedure are available, either (1) enabling the lung to expand to fill the cavity by removing its restricting membrane of exudate or (2) allowing the chest wall to collapse to meet the lung. The first of these which is comprised in the Fowler-Delorme type of operation has already been briefly discussed. The second method is employed in operations of the type of Estlander, Schede, etc. It is chiefly this second method which will be discussed here.

Disadvantages of Collapsing Operations

Collapsing operations have many important disadvantages, as every surgeon well knows. These operations are accompanied by so high a mortality that from this standpoint they constitute some of the most formidable in the surgical repertory. They are always very mutilating in character, and they do not by any means always result in healing, even if the patient survives the immediate risk of death from the operation itself.

The extent of the mutilation induced by these operations has received almost no attention. The anatomic mutilation with its very bad cosmetic effect is of course readily apparent. The physiologic deficiencies, however, which are probably made permanent by these operations, I have been unable to find discussed anywhere. It is this latter phase which has particularly interested me, and recently the opportunity has presented itself for a study of this aspect. Typical results are illustrated in the following case.

A young unmarried woman, a nurse, aged 28 years, recently came under my care because of a small discharging sinus in the left side of the thorax. She stated that in the winter of 1918 she had a bronchopneumonia followed by an empyema of the left side for which she was operated on by the resection of a portion of one rib while still very ill. No irrigations of any kind and no suction apparatus was used. She continued to drain, and about three months later a partial Schede operation was done which consisted in the extensive removal of four ribs. This failed to result in healing and two months afterwards (about June, 1919) two or three more ribs were removed. Following the last operation she was treated sporadically with what was said to be "Dakin's solution." Still, however, she continued to drain pus. At the time of my examination it was observed that from four to six inches of each of the ribs from the third to the tenth had been removed from the lateral aspect of the left thorax. The usual apparent deformity resulting

from such a procedure was present. In the mid-axillary line at a level corresponding about to the sixth rib there was a small sinus from which pus issued. When a probe was inserted into the sinus it was found to pass upwards and inwards for a distance of about three inches, and when an attempt was made to irrigate the sinus a communication with the lung was discovered which resulted in coughing and in expectoration of the fluid. She was nearly forty pounds under her normal weight, and she had a temperature which reached 101° nearly every afternoon. She was also exceedingly morose both because she was unable to work properly and also because her deformity worried her greatly. The pulmonary fistula was closed by another operation after which the systematic use of Dakin's solution was carried out. On her discharge from the hospital after two and one-half weeks her temperature was continuously normal, she had gained about five pounds in weight and the wound was healed except for a small angle of the skin through which the tube had passed.* The chief matter of interest, however, in this connection is that measurements of her vital capacity both before and after operation gave an average reading of only 1490 c.c. with a Krogh spirometer. The highest individual reading was 1625 c.c. and the lowest was 1375 c.c. There was no material difference in the readings before and after her slight operation of closing the pulmonary fistula. Her height was 5 feet, 7 inches. When we compare this value with the table of vital capacities for normal women, as given by Peabody and Wentworth, it is apparent that instead of having a value of 3275 c.c., which is the normal average of women of her height, she had only about one-half of this capacity. It is interesting also that, as is usual, in spite of her diminished vital capacity, the readings for the tidal air varied between 400 c.c. and 500 c.c. and were therefore normal.

*I have since learned that after remaining closed for about two months the sinus opened again.

Collapsing Operations Should Become Rare

The importance of this marked reduction of vital capacity in this patient after having had a Schede operation is very great; for it means that, despite the fact that her empyema has apparently healed, she is left in so mutilated a condition that in all probability if she ever has pneumonia, a pleural effusion, a disturbance of her heart, or any other condition which lowers the vital capacity she will have only about a 50 per cent chance of recovery as compared with what she would have if her vital capacity were normal. Besides this probably permanent physiologic defect, she has a bad anatomic deformity, she is almost a psychopathic patient, and she has practically lost a year. It is felt that the application of the leading principles which are under discussion in this article will practically prevent the necessity for these mutilating operations. It is remarkable how the intelligent use of Dakin's solution, combined with proper drainage and with suitable exercises to promote expansion of the lungs, such as blowing against resistance, calisthenic exercises, etc., will obliterate a cavity in the chest which is so large that obliteration without operation seems hopeless. This fact has impressed itself forcibly upon all who have used these methods intelligently and conscientiously. I have seen, for example, a cavity of a capacity of 1500 c.c. which had remained stationary for three months become entirely obliterated with permanent healing by the application of these methods. This fact is forcibly emphasized in the report of the Empyema Commission, "the increased expansion of the lung following the use of Dakin's solution leads to the hope that extensive intrathoracic operations, such as decortication of the lung, may in most cases prove unnecessary. Such procedures should be undertaken only after most prolonged efforts to obtain expansion of the lung have failed." In the great majority of cases which fail to respond to this treatment some other

factor than an ordinary empyema will be found responsible for preventing the healing, such as the presence of a foreign body, or tuberculosis.

The closure of empyema cavities after sterilization has been accomplished, despite the presence of a pneumothorax, has been recommended by Stewart,[24] Tuffier,[25] Moschcowitz,[26] Heuer[27] and others. This procedure will sometimes yield brilliant results but in many instances, in the majority in the opinion of the present author, the cavities will fill up with fluid and become reinfected. One should not be too optimistic in regarding a case as healed merely because months or even a year have passed without symptoms. If followed long enough, many of these cases will show recurrences. I have had one case in which after the patient had been well enough to perform the duties of brakeman on a freight train for a year I reopened his chest and obtained more than 1000 c.c. of pus containing a streptococcus. Available methods of determining the sterility of the cavities are not sufficiently reliable to establish with certainty the question of sterility of the tissues themselves which compose the walls.

SECTION III

CAREFUL ATTENTION TO THE NUTRITION OF THE PATIENT

In some studies on the metabolism of patients with streptococcal empyema, R. D. Bell, of the Empyema Commission, made some observations which are of very great importance. These are published in the report of the Empyema Commission to which a reference has already been made. Briefly he found that unless special attention is paid to the nutrition of these patients they are likely to have a negative nitrogen balance amounting to a deficit of as much as 21 grams per day. A negative nitrogen balance was always found if the diet consisted of only 1500 to 1700 calories per day. If a patient is losing in his excretions 21 grams of nitrogen per day more than he is taking into his body as food, it is not surprising that he loses rapidly in weight. Obviously if this negative nitrogen balance continues very long the disadvantageous effects of starvation are added to the handicaps under which he is already laboring in his efforts to combat a serious infection. If, however, the food intake is pushed so that he has a mixed diet containing from 3300 to 3500 calories per day, his nitrogen balance will be positive and he will usually gain in weight instead of lose. It is very interesting in this connection that Bell found that the average daily loss of nitrogen in the pleural exudate in patients who were aspirated amounted to only about 2 grams, as compared with from 20 to 30 grams in the urine.

The advantages of preventing loss of weight and emaciation are so obvious that they need no comment. Emphasis should, however, be placed on the fact that it is perhaps as important a duty on the part of the surgeon to see that

these patients are taking a sufficient quantity of food as to attend to any other part of the treatment of an empyema. Undoubtedly many fatalities and chronic cases are due to the neglect of this important feature. Difficulty in persuading patients to take a sufficient amount of nourishment is usually encountered only during the period in which they have fever. With the subsidence of the fever the appetite usually returns promptly. But even during the febrile stage a very considerable amount of nourishment can be given to patients easily by the administration of liquids. A particularly effective way to get large amounts of carbohydrate into such patients is by the administration of drinks containing lactose, since this sugar is not sweet and has so little taste that it may be easily disguised by the presence of fruit juices, etc. Woodyatt[28] has also suggested the subcutaneous administration of a 2 or 3 per cent solution of glucose which I have used to advantage in some cases. The intravenous method of administering it is of course well known. The importance of keeping the body well supplied with carbohydrate in cases of acute infection is now generally recognized, and much of the striking detoxicating power of the liver is at the present time ascribed to its content of glycogen. Opie and Alford[29] have found that in general those substances which have a particular tendency to produce lesions of the liver are markedly inhibited in their action by carbohydrate feeding. The present author also in 1915[30] showed that the peculiar insusceptibility which Whipple[31] had found young pups to possess against the production of late chloroform poisoning was in reality due to the high glycogen contents of their livers. As has also been shown by H. T. Graham and the author[32] it is possible that at least some of the protective action of glycogen may be due to the fact that in its depolymerized form (glucose) it retards the diffusion of acids and other substances through gels. Its protective action

and antiketogenic effect in conditions of acidosis is well known.[33]

As a part of the discussion of the importance of maintaining the nutrition, mention may be made also of the necessity of keeping up the water balance of the body by the liberal administration of fluids. Recently a new significance has been given to this question by the important work of Balcar, Sansum and Woodyatt.[34] These authors present striking evidence in favor of the view that the presence of fever in the various infectious diseases may be due to an insufficient amount of "free" water in the body, that is of water which is not bound by physical or chemical union to various substances in the body, and they suggest that high fever in disease may often be markedly reduced by the administration of sufficient amounts of water.

SECTION IV

DISCUSSION

As stated in the introduction, particular emphasis in this article has been placed on a consideration of what seem to be the cardinal principles involved in the treatment of empyema rather than on details of treatment. Details have been mentioned and discussed only when it seemed advisable to do so for the purpose of more easily establishing the validity of the principles involved.

Fistulas Which Communicate with the Lung

There are, however, certain questions of the nature of details which perhaps merit some discussion. In the first place, there is the question of the method of handling cases in which there is a fistula communicating with the lung. Often these cases cannot be adequately treated with Dakin's solution or by any other method of irrigation because of the discomfort and possible danger to the patient induced by allowing the entrance of the fluid into the lung. Experience has shown that a safe way of handling these cases is by temporarily avoiding irrigations but allowing for free drainage and keeping up the nutrition and body fluids as well as possible. The majority of the small openings close spontaneously and give no serious trouble. After they have closed, the use of Dakin's solution may be commenced. Although surgical closure of such fistulae can often be accomplished, yet it should be remembered that when present they indicate an intrapulmonary suppurative process and are often safety valves for the discharge of pus from an abscess. Premature surgical closure therefore is not only unsuccessful but it may often be disastrous. It is wise

to allow an interval of at least several months to elapse before attempting an operative closure.

When Is an Empyema Healed?

Another question which arises is, when may an empyema be considered healed? In another section it has already been stated that a case of empyema cannot be considered as healed until the cavity has been both sterilized and obliterated. It is clear then that we must be sure that sterilization has occurred before allowing the cavity to close, and the only way of determining this is by making cultures of the wound secretion at frequent intervals. It is always dangerous to allow the external opening to close if there are any streptococci still present in the wound, and it is unwise to allow it to close unless at least two or three successive cultures have shown no growth. Many startling new methods of treating cases of empyema have been introduced based on extravagant claims of incredibly speedy healing. These methods usually, instead of being new, are merely new combinations of well-known details, and their authors frequently are apparently unaware of or ignore the well-known fact that to nothing does the old proverb of "haste makes waste" apply with more truth than to the treatment of empyema. In the ordinary case there is no difficulty at all in accomplishing the rapid closure of a small drainage opening; and it is surprising that a patient may frequently go around in a fair degree of comfort for several months with a gradually accumulating lake of pus in his chest. But merely because his external wound is closed and merely because he complains of no discomfort is not sufficient evidence upon which to conclude that he is permanently well. All cases of empyema which have closed should be watched most carefully for at least six months and subjected to frequent rigorous examination, both by

ordinary means and the x-ray. If doubt arises about a reaccumulation of pus, an aspirating needle should be inserted. Many cases have been deliberately closed surgically after assurance has been given that sterilization has occurred, even if a cavity of moderate size remains. It not infrequently happens, however, that such cases have to be reopened, and although many are entirely successful, a decision that in a particular case permanent healing will occur must be reserved until at least several months later.

Adequate Drainage Desirable in Subacute and Chronic Stages

The question of methods of drainage has been only lightly touched on in this article. It is felt strongly, however, that after the acute pneumonic symptoms have subsided care should be taken to see that the drainage is adequate, regardless of whether it is accomplished by an open method or under negative pressure. Most of the chronic cases which have come under my personal observation have had very inadequate drainage, and I have been impressed with the idea that in many the chronicity was due very largely to the inadequacy of the drainage. It is unfortunate that the very striking effects of Dakin's solution in empyema have led some surgeons to rely entirely on this agent without providing for efficient drainage. In an experience with 138 chronic cases at Fort Sheridan, Illinois, during the summer of 1919 it was common to see men arrive who had had empyema for from six months to a year with no provision for drainage except a snugly-fitting tiny catheter or a Carrel tube the end of which often would be tied in a knot and thus drainage would be effectually prevented. I feel that the combination of the intelligent use of Dakin's solution with adequate drainage is nearly an ideal one, but either one alone accomplishes little.

No Discord between Experimental Results on Pneumothorax and Clinical Findings in War Wounds of the Thorax

Because of the experience of many surgeons at the front which revealed the fact that open wounds of the thorax, often of amazing size, were not necessarily fatal, a belief among some has become prevalent that there is therefore no danger from an open pneumothorax. As a result of this belief some of the experimental results on pneumothorax which have been described in this article have been criticised. Sir Berkely Moynihan,[35] for example, in discussing the open operation for extraction of foreign bodies from the lungs and pleural cavities states that "no shock follows this operation," also, that during the operative incision of the pleura the free admission of air, "as a rule, causes no disturbance and does not alter the rate of the respirations or of the pulse." He bases his remarks chiefly upon articles by Duval, Depage, Gregoire, Elliott, etc., but he says practically nothing about how extensive his own experience with wounds of the lungs and pleura has been. On the other hand, reference to the work of Duval[36] and of others who have had extensive personal experience with this type of cases gives one a very different impression from that obtained from Moynihan's article, although at their hands also results were obtained which seem startling when compared with our ideas of the limitations of thoracic surgery before the war. For instance, Duval's article, which is based on 3453 collected cases, shows that of all types of wounds of the chest the mortality is nearly twice as great in the gaping as in the closed wounds (27 and 15 per cent respectively). The larger mortality of the gaping wounds is ascribed by Duval chiefly to the element of infection, but it seems highly probable that many of these may have died directly of the open pneumothorax, especially since, according to Duval, 50 per cent of all lung wounds die within the

first day, before the results of infection could have manifested themselves. Certain ones of course die of hemorrhage. Piéry states that the two gravest prognostic elements are the large open chest wound and generalized infection. "In[37] discussing operative indications, Piéry sounds a moderate note of conservatism when he points out that he considers it unfortunate that the early optimistic reports regarding the safety of lung surgery must in a measure be modified—in other words, one must bear in mind that surgery of the lung, in spite of the remarkable recent advance, is still surgery of a very grave sort." Both Duval and Piéry consider that the gaping wound must be closed in order to overcome the resultant mechanical embarrassment of respiration and the resultant pleural infection. Indeed, according to them, closure is to be considered as an emergency operation.

As regards the operative attack on the lung itself it is to be noted that most surgeons with wide experience in war wounds of the lungs adopt measures which of necessity tend to limit the actual area of the opening into the pleural cavity and also to immobilize the mediastinum somewhat. In other words, they follow, either consciously or unconsciously, methods which our experiments show to be effective in reducing the danger arising from an open pneumothorax. For example, as a rule, the lung is drawn out through the thoracic incision, a procedure which not only diminishes, if it does not actually plug, the opening into the pleura, but also tends to immobilize the mediastinum. Moreover, frequently the thoracic opening is plugged by a thick gauze compress after the delivery of the lung in order to obviate the to-and-fro movements of air during the operation. "It[38] is necessary to operate as gently and quickly as possible and to get the opening in the chest wall closed at the earliest possible moment. In case there should be any evidence of mechanical interference with breathing it is advisable rapidly to deliver the lung, plugging the chest

wall with gauze. This procedure is usually followed by cessation of all respiratory embarrassment." Duval says that the closure of the chest wall must be done very carefully in order to avoid the leakage of air; the resected rib ends should be covered with muscle in order to get an air-tight wound. "Although the operation is described as sometimes remarkably simple it is, nevertheless, exceptional for these patients to run other than a rather stormy post-operative course." Roberts and Craig[39] in a report based on 199 cases state that they operated upon "open" cases as soon as the condition of the patient permitted and that before following this method the great majority of cases never reached the base. Anderson[40] in an experience in a British casualty clearing station found that the closure of an aperture in the wall of the thorax more than compensates for any risk which is accepted, and he emphasizes that the important matter is to get the chest completely closed. Gask and Wilkinson[41] in a report based on 500 cases of this type, also at a British casualty clearing station, adopted the routine of always closing large openings into the pleura, through which air was sucked, by temporary skin suture without an anesthetic. This was found to give the patient immediate relief.

It is clear, therefore, that from the experience of many surgeons in a very large number of cases of war wounds of the thorax the dangers of open pneumothorax cannot be disregarded. It is not surprising, however, that otherwise healthy men can stand during the period of an operation a relatively large opening in the thorax, for in a previous section of this article it is shown that from calculations based on experiments, a healthy adult of average size, without pneumonia and with good respiratory muscles, should be able to stand for a short time without death an opening of at least 51.5 sq. cm. or 8.1 sq. inches. It is shown, moreover, on the basis of the same calculations, that individuals whose vital capacities are above the average will be able to

withstand larger openings; thus, for example, the man with the vital capacity of 7180 c.c. ought to be able to endure an opening of about 101.3 sq. cm., or about 15.6 sq. inches as long as his respiratory muscles are able to effect a maximum compensation. The openings here given are considerably larger than the usual area of an opening into the chest during the course of an operation, because such an opening is nearly always actually much smaller than it seems to be owing to the presence in the incision of a delivered lung, fingers of the operator or assistants, gauze pads, instruments, etc. There is, therefore, no lack of harmony between the conclusions drawn from the experiments on pneumothorax and the clinical observations on war wounds of the chest. The application of these results to thoracic surgery in general is obvious.

The striking mobility of the mediastinum has been studied with the x-ray by Stivelman and Rosenblatt[42] in cases of artificial pneumothorax. Although the pressures in the two pleural cavities were not measured, the reproductions of x-ray plates illustrate so clearly the mobility of a mediastinum which is not fixed by adhesions or induration, that apparently these observations afford some direct confirmation of the experimental results on pneumothorax.

SUMMARY AND CONCLUSIONS

Empyema in previous years has been one of the most serious problems in surgery, as judged by the mortality figures alone. The opportunities for the study of a large number of cases under favorable conditions in the military hospitals have resulted in modifications of treatment which give promise of not only greatly reducing the mortality but also the disability from this condition.

The essential points of these new principles of treatment are: (1) Careful avoidance of open pneumothorax in the acute, pneumonic stage, (2) the prevention of a chronic empyema by the rapid sterilization and obliteration of the infected cavity, and (3) careful attention to the nutrition of the patient.

Experimental work on pneumothorax, discussed in detail above, shows that previous conceptions of the mechanics of pneumothorax are erroneous. These conceptions have been based on the false idea that the mediastinum constitutes a more or less rigid partition between the two pleural cavities in the normal individual, and they have led to the belief that when an open pneumothorax is established the lung on the same side is collapsed and respiration is maintained by the other lung.

On the contrary it has been shown by experiment on the human body that the mediastinum offers only a slight resistance when the pressure in one pleural cavity is made equal to that of a column of water 10 cm. high. Experiments with greater pressures than this have not been carried out.

In the normal individual there is at all times practically an equilibrium of pressure throughout the whole thorax, so that when an open pneumothorax on one side is created

both lungs are compressed and respiration is probably maintained by both lungs.

Obviously factors which tend to immobilize the mediastinum, such as adhesions and thickening from long-standing inflammation change these relationships by making of it a more rigid partition.

The maximum opening into a pleural cavity compatible with life depends upon a definite relationship which exists between the amount of air entering the lungs and the amount which enters the pleural opening. This may be approximately determined for the normal chest by the mathematical expression given in the text. By this expression it is found that an *average* normal human adult should be capable of withstanding for a short time an opening of about 51.5 sq. cm. (5×10 cm., or 2×4.1 inches), but in exceptional instances this value may be very much larger, depending to a considerable extent on the vital capacity. As in one instance, mentioned in the text, it may reach the very high value of 101.3 sq. cm. (15.6 sq. inches). The length of time that life will be maintained in the normal individual with a maximum opening will be determined largely by the strength of his respiratory muscles.

The bearing of these results and deductions upon both the treatment of acute empyema and upon thoracic surgery in general is obvious. Whenever the amount of air taken into the lungs is limited by the presence of an active pneumonia, with plugging of both air channels and alveoli, whenever there is an excessive demand for air, whenever there is sufficient weakening of the respiratory muscles to impair compensation or, in short, whenever the vital capacity is reduced, the size of a pleural opening compatible with life becomes smaller; and if any or all of the above factors are present in sufficient intensity, even a very small opening into the pleural cavity will produce death from asphyxia. Since all of these factors are likely to be present to a high degree during the early stage of an empyema of

the streptococcus type, early operation with the establishment of an open pneumothorax carries with it an unwarrantable danger.

Other important effects of an open pneumothorax are rapid heat loss, danger of infection and disturbance of the systemic circulation.

The cardinal objects in the treatment of empyema are sterilization and obliteration of the cavity. Unless both of these objects are attained permanent healing does not occur.

Dakin's solution (neutral 0.5 per cent solution of sodium hypochlorite), when intelligently used, and combined with adequate drainage, is able to accomplish both of these objects simultaneously. As a decorticating agent it has proved so satisfactory that the belief seems justified that the necessity for the performance of mutilating operations of the type of Fowler-Delorme, Estlander, Schede, etc., will almost never arise, particularly in those cases which have been treated properly from the beginning.

Operations of the type of Estlander's and Schede's result in probably a permanent marked reduction in the vital capacity, besides being associated with a high immediate mortality.

A marked loss of nitrogen occurs during the acute stage of a streptococcus empyema, and, therefore, careful attention should be paid to the maintenance of the nutrition of these patients. The maintenance of an adequate supply of water is also important.

The experimental results on open pneumothorax seem to harmonize satisfactorily with the clinical findings on war wounds of the chest.

ADDENDUM

The long delay in the publication of this essay since it was written has been associated with the appearance of several articles by various authors which have contained conclusions at variance with some of my experimental observations and conclusions on pneumothorax. There is, for example, a frequently quoted statement in the literature that the dog differs radically from the human in that normally there is a communication between the pleural cavities of the dog, while in the human there is no such communication. This statement has been made by Matas,[43] Duval,[44] Yates,[45] and others. More recently Synder[46] has claimed that it is impossible to produce a unilateral pneumothorax in the dog because of the alleged communication between the two pleural cavities. I have examined 25 dogs with the particular purpose of finding out whether there are any anatomic openings in the mediastinal pleura and have never found any. It is interesting also that very recently Duval[47] and Matas[43] have retracted their former opinions about the presence of a normal communication between the pleural cavities of the dog. Synder[46] has stated that in the dog it is impossible to create a unilateral pneumothorax because the air passes at once from one pleural cavity through the mediastinum to the other one with the result that the pneumothorax is bilateral. He shows also that fluids pass readily from one side to the other, and he quotes Garland[48] and Calvert[49] as having previously called attention to this fact. Both Duval[47] and Matas,[43] although now admitting the absence of any anatomic communication between the two pleural cavities in the dog, nevertheless maintain that in this animal the mediastinal septum is so thin and tenuous in comparison with that of the man that experimental observations of pleural pressures made in the dog are not

applicable to the human thorax. In our original article in 1918 we had concluded that they were applicable in all particulars. In reply to this phase of the controversy I wish to state that I have repeatedly produced a unilateral pneumothorax in the dog with marked deflection of the heart and mediastinal structures into the opposite pleural cavity and I have published the confirmatory x-ray reproductions.[50] A reopening of the study, however, has convinced me that in many dogs the pneumothorax does soon become bilateral and that there is a considerable difference between even apparently normal dogs in the amount of resistance to pressure offered by the mediastinum. In some dogs simultaneous readings of the pressures in the two pleural cavities after the injection of air into one give results very similar to those in the human; but in others the results are such as would be expected in a bilateral pneumothorax. Even if one concludes that the dog may not be an animal entirely suited for the experimental study of *unilateral* pneumothorax, it would seem nevertheless that this animal would be suitable for the study of the phenomena associated with *bilateral* pneumothorax and that the results obtained in such a study would be applicable to a consideration of bilateral pneumothorax in the human. Even if one admits that the mediastinal septum in the dog is more easily stretched, more easily torn and more easily penetrated by gases and fluids than is the human, it is nevertheless perhaps not out of place to call attention to certain irregularities in the methods of experimentation. If one wishes to measure the pressure in the opposite pleural cavity after injecting air into one, it is necessary to permit the entrance of a little air into the opposite cavity in order to have a cushion of air around the cannula which will permit a reading. If this is not done, the lung will usually force itself against the end of the cannula and prevent any oscillation of air within the cannula and tube. One starts therefore with some degree of bilateral pneumothorax

which probably results in the retraction of the lung away from the mediastinal pleura. The final result is that if the lung is retracted away sufficiently the mediastinal septum will be hung in space at its edges without the buttressing support of the lung against it. It is obvious then that a thin mediastinal partition will be ruptured more easily under such circumstances than under the conditions which are presented in the case, for example, of a surgical pneumothorax. For in the latter the only air which can be present in the opposite pleural cavity will be that which may have diffused through the mediastinum; moreover the mediastinum will be everywhere supported by being buttressed against the opposite lung. What we are really desirous of knowing, therefore, is not merely what the resistance of the mediastinal partition is but also how much is the other lung compressed by a force transmitted through the mediastinum. Nearly all of the experimental observations that have been published are really concerned only with the question of the resistance of the mediastinum.

Even the admission on my part that experimental observations on unilateral pneumothorax in the dog may not be applicable to man in their entirety does not in any way annul the chief conclusions to be drawn from our earlier work, although these have been severely criticized by Duval.[44] He states, for example, that the size of the pleural opening is not a matter of great importance as regards the possibility of causing death. He is a strong advocate of a very large incision for the expressed purpose of making a total unilateral collapse of the lung to permit no respiration at all on that side and no "ventilation" of the pleura. He desires to make that lung completely airless. He states that this procedure also prevents the circulation of "pendelluft," a term used by Brauer to indicate the theoretical transfer of air from the unaffected lung into the partly compressed lung at expiration and the rebreathing of this air into the uncompressed lung on inspiration. Brauer's idea

was of course that this phenomenon was an important factor in the production of asphyxia because this air would have its oxygen diminished besides containing an excess of CO_2 and would also prevent the inspiration of that much fresh air. Duval claims that the thorax of the bovine class of animals resembles that of the human and that an animal of this class should be used for experiments instead of the dog. He states that an experiment which he performed on a calf lends support to his ideas. He considers that in order to make a very large opening safe all that is necessary to do is to immobilize the corresponding side of the chest and diaphragm by means of a powerful automatic retractor and to express from the lung its residual air. To accomplish this he delivers the lung out of the wound. He also recommends enveloping and progressively compressing the lung with warm gauze compresses. He regards the "ventilation" of the pleura as the most serious and harmful factor resulting from an open pneumothorax. By this he means presumably the passage of air in and out of the pleural cavity. In his criticism of our work he makes no reference at all to the importance of the vital capacity of the patient, a point which we strongly emphasized. I[51] have already replied to Duval's criticisms in a separate article; but I shall discuss some of his points and conclusions here.

It is rather generally admitted that a unilateral open pneumothorax may sometimes have disastrous results. Duval's conclusions differ diametrically from mine as to the reason for these disasters. He considers them as being due to the fact that the opening is not large enough, whereas on the contrary, the evidence seems to me to point to the fact that they arise because in a given case the opening has been too large. From the practical standpoint of surgical methods it is of the utmost importance to settle this question definitely. For that reason it will be well to analyze with some care the effects of a pneumothorax in order to gain a clearer insight into this important question.

The majority, if not all, of investigators who have used animals for the experimental study of pneumothorax have observed a change in the character of the respirations after the creation of an open pneumothorax. Likewise most surgeons of wide experience in thoracic work whose observations have been carefully made have noticed a change in the character of the respirations in the human in the presence of an open pneumothorax. Usually the first alteration is an increase in the depth of inspiration (an increase in amplitude), followed later by an increase in the rate. In an earlier paragraph I have already pointed out that the act of getting air into the lungs consists chiefly of enlarging the thorax, a procedure which necessarily reduces the intrapleural pressure (makes it more negative and thus causes air to rush down the trachea to inflate the lungs). We should expect similarly that whenever a sudden interference with the intrapleural pressure occurs which would make it equal to that of the atmosphere, the natural compensatory mechanism to offset this would be an attempt to create again a negative intrapleural pressure in order to get air into the lungs. The simplest way to accomplish this is to enlarge the thorax still more in an effort to reduce its interior pressure. This is just what happens then as an automatic effort to counteract the sudden increase in the pleural pressure. This reveals itself in an increase in the depth or amplitude of the respiration. For purposes of simplicity we can compare in this respect the lungs and pleural cavities with any object containing gas, as for example, a toy balloon. If the balloon should suddenly be doubled in size, or if the gas were suddenly transferred to a balloon of twice the size of the original one, the pressure of the contained gas would be only one-half of what it had been originally. In a structure like the thorax which is constantly changing its size, an opening communicating with the outside air does not necessarily imply that the pressure within is always equal to that of the atmosphere, especially if the opening

is a small one. This has been a common source of confusion. During inspiration the intrapleural pressure is usually negative and during expiration it is positive, if the opening is not too large.

Haldane and Poulton[52] have shown that if an individual is placed in an atmosphere containing 4 to 5 per cent of CO_2 his respiration increases in depth and little by little increases in rapidity. On the other hand, if the amount of inspired oxygen is suddenly reduced below 14 per cent there is also at first an increase of amplitude of respiratory movements which they think is to be attributed to the fact that the anoxemia, by lowering the threshold of the respiratory center for CO_2 provokes the expulsion of CO_2 from the body. At the same time the rate is accelerated as the result of the anoxemia. Finally the respiration becomes periodic. If one continues to reduce the percentage of oxygen in the inspired air to 10 per cent the respiration becomes superficial and finally death occurs. Haldane, Meakins and Priestley[53] more recently have shown that if, instead of lowering the partial pressure of oxygen in the inspired air, one leaves the air at its normal composition but diminishes the amount of air entering the lungs sufficiently to produce a superficial respiration, the respiration soon becomes periodic. Periodic respiration is known always to be due to lack of oxygen. The anoxemia therefore induces superficial respiration which itself maintains the state of anoxemia. A serious vicious circle is therefore established.

Now Duval states that in a unilateral open pneumothorax dyspnea exists as long as the corresponding lung respires partially and "danses" a little in the thorax, that is, as long as the external air strongly ventilates the pleura, but that as soon as that lung is put into condition of complete collapse the dyspnea disappears. I have not been able to confirm this observation although, in accordance with Duval's recommendation, I used a calf for experiment. Moreover Dautrebande and Spehl[54] fail to agree with Duval in spite

of the fact that they used rabbits, animals which, like the bovine group, Duval recommends for experimental use because of their similarity to the human. In fact, in direct contradiction to Duval's claims I found that a calf dies if a large unilateral opening is made in the chest. It is difficult to understand how there could be any disagreement on a point so easy to demonstrate experimentally. When a large opening is made, however, the respirations soon become much more superficial, a fact which perhaps Duval has interpreted as a reduction in the dyspnea. But this is not to be regarded as a favorable sign. On the contrary, as Dautrebande and Spehl have shown in their excellent article, it should be looked upon as a danger signal because it indicates an anoxemia. This conclusion would agree very well with the work of Haldane and his pupils to which reference is made above. Accordingly Dautrebande and Spehl state that they regard Duval's conclusions as erroneous.

As has been mentioned above, Duval regards a large opening in the chest as less dangerous than a small one. My own opinion, however, based on many experimental and clinical observations is that, other things being equal, the larger the opening the greater the danger to life. The reasons for this conclusion are many. In the first place, it has been a frequent observation that animals will endure a bilateral open pneumothorax almost indefinitely if the openings in each side of the chest are not too large. In the case of dogs and cats of ordinary size, for example, if a small trocar is inserted into each pleural cavity the animal will go on for hours with often no evidence of any disturbance except for an increase in the amplitude of the respiratory movements, and this disturbance will promptly disappear after the withdrawal of the trocars. If, however, too large openings are made the animal will soon die with the picture of asphyxia. It seems to me that this simple experiment permits several important conclusions. It shows

first of all that a lung does not become functionless merely because outside air is allowed free entrance to the pleural cavity. It shows also very clearly that the important consideration is the size of the openings. Other subsidiary factors in the production of death, to which great importance has been attached by some, such as "pendelluft" and the fluttering of the mediastinum, are ruled out in a case of bilateral open pneumothorax, induced by openings of the same size, which has proved fatal; for in such a case, since the pressures are equal in both pleural cavities at all times, there will be no to and fro motion of the mediastinum and also no chance for the occurrence of "pendelluft," and yet the animal dies. Now when we come to consider the question of a unilateral open pneumothorax we find that practically the same phenomena occur. The principles involved are substantially the same as are involved in a bilateral opening because the effects of a unilateral opening are not confined to only one lung but concern both lungs. The details, however, are slightly different, because, as has been said above, the pressures in the two pleural cavities during a unilateral open pneumothorax are not quite the same and because there is some to and fro movement of the mediastinum, etc. Nevertheless, in view of what we have just said about a bilateral open pneumothorax, it seems reasonable to suppose that with a unilateral opening such questions as the fluttering of the mediastinum and the action of "pendelluft" are of less importance than the direct effects on the lungs caused by the altered pressures. This brings us back again to the importance of the size of the opening.*

*In a recent article Lundsgaard and Van Slyke[65] invoke an idea similar to that of Brauer's "pendelluft" as an explanation of cyanosis in unilateral open pneumothorax. They quote experiments by Sackur[66] who, after producing a supposed collapse of one lung in rabbits and in a dog by unilateral open pneumothorax, found a diminished content of oxygen in the arterial blood. On the basis of these experiments they conclude that cyanosis in acute unilateral open pneumothorax is produced "in the same manner as when the air passages to one lung are blocked, viz., by blood flow through unaerated lung space." In considering the pressure effects of open pneumothorax as if only one lung were concerned, they show that they are unaware of the fact that these effects involve both lungs. This leads them to the assumption that the frequent absence of cyanosis in spontaneous pneumothorax in tuberculosis is due perhaps to extensive thrombosis of the vessels in the lung on the involved side, a condition which they infer would prevent cyanosis because

The usual attempt to compensate for the increased pleural pressure is to take a deeper inspiration, as has been discussed above. Theoretically all that is necessary to do to prevent a fatal asphyxia is to get enough air into the lungs to satisfy the tidal air requirements. This can be accomplished of course in spite of an open pneumothorax provided that too much air does not enter the pleural cavity with each inspiration. If so much air enters the pleural cavity through the open pneumothorax wound that pulmonary inflation is sufficiently compromised to prevent the possibility of inhaling enough air into the lungs to maintain life, then of course a fatal asphyxia will occur. Fortunately here, as in other respects, nature has made provision for a generous reserve. The tidal air requirements ordinarily constitute only about one-eighth to one-twelfth of the total amount of air which can be inhaled if a maximal effort is made. This latter amount of air represents what is known as the vital capacity. It is apparent therefore that a very considerable amount of lung tissue can be made functionless and yet permit the individual to take in enough air to maintain life by increasing his respiratory movements. This then would seem to be the explanation of the well-established fact that life is compatible, in a vigorous adult, with a relatively large unilateral opening and even with bilateral openings whose combined areas greatly exceed the area of the opened glottis. A common error in the older literature on pneumothorax was that a lung becomes collapsed and functionless if a pleural opening as great or greater than the diameter of the glottis is made. A person who cannot make so great a respiratory effort cannot resist so large a pleural opening as the one who can make a great inspiration; likewise it becomes apparent that the same individual, if he

a relatively small amount of blood would pass through that lung. It seems to me just as logical to assume that the explanation of the absence of cyanosis in such a case lies in the fact that because of the presence of adhesions and fixation of the mediastinum there are no serious pressure effects on the opposite lung and that therefore actually a less amount of lung tissue is involved than if no adhesions were present, to say nothing of the altered relationships of the heart and large vessels when a pneumothorax is induced in the absence of adhesions.

has any serious impediment to his respiration, will die from a pleural opening which would not be fatal if the impediment were absent. In other words, therefore, the vital capacity of the individual is of great importance in determining the size of a pleural opening which is compatible with life. One who has a large vital capacity can withstand a larger pleural opening than one whose vital capacity is low. By considering what conditions lower the vital capacity we can understand readily that a large pleural opening is likely to be fatal in an individual whose respiratory muscles are weak, who has an uncompensated heart, who has an extensive pneumonia, a pulmonary edema, an acidosis, a deep general anesthesia, etc. Indeed, if he is so dyspneic that he just succeeds in maintaining life in spite of maximal respiratory effort, in other words if his vital capacity practically equals his tidal air requirements, he may succumb to the effects of even a very small pleural opening.

In a previous section I have called attention to the fact that on purely theoretical grounds, even if an alteration of pressure in one pleural cavity induced a change in the other of the same degree it can be calculated that a pleural opening can be withstood of such a size that it would have seemed impossible before the war experience. For example, on the basis of our calculation, a man with a high vital capacity (7180 c.c.) could support life with a unilateral opening as large as 15.6 square inches, or 101 square centimeters. These calculations, however, are not strictly accurate but are only approximations because of the variability of several factors. As a matter of fact, since the pressures in the two pleural cavities are not equal when there is a great increase in one, it is seen that in some individuals with high vital capacities a unilateral opening considerably larger than 101 square centimeters may be compatible with life. It really is of little importance to know exactly in any given case how large an opening can be made. The im-

portant thing to realize is that those whose vital capacities are low, especially those who are already dyspneic, may die as a result of even a small opening unless measures are taken to prevent serious consequences.

The most important aid in minimizing the harmful effects of an open pneumothorax is to close the opening. Closure of the opening leads to an almost instantaneous improvement in the respiration. The explanation of the relative innocuousness of a closed as compared with an open pneumothorax is apparently as follows: When an opening is closed, particularly if it is closed at the end of expiration, there is not enough air remaining in the pleural cavity to encroach on the breathing surface of the lungs sufficiently to prevent the establishment of a negative intrapleural pressure by a slight increase in the depth of inspiration. There is no longer an active competition between the passage of air down the trachea on the one hand and the entrance to air into the pleural cavity on the other hand. The impossibility of getting the requisite amount of tidal air into the lungs does therefore not occur. Experimentally, however, a closed pneumothorax can be made to cause a fatal asphyxia if enough air is injected into the pleural cavity. Here again the process seems to consist fundamentally of increasing the intrapleural pressure to such an extent that it becomes impossible to create a negative pressure in spite of maximum respiratory efforts; the necessary amount of tidal air cannot be obtained, with the inevitable result that asphyxia occurs. Again, moreover, if the vital capacity is low, it is easier to produce death with a closed pneumothorax for reasons which have been made sufficiently clear in the discussion given above. Doubtless some of the catastrophes which have occurred in the production of artificial pneumothorax for therapeutic purposes are explainable on the fact that patients with low vital capacities and without adhesions

have been given too much air. An amount of air which might be well tolerated by a patient with a high vital capacity might be fatal in one whose vital capacity is low. The prompt withdrawal of air in an alarming case will usually improve the dyspnea and prevent death. It is an interesting fact that Duval and others who have criticized our theory of the action of pneumothorax recommend strongly the use of measures which tend to reduce the size of the opening when performing operations on the chest. They therefore unconsciously support our theory while at the same time they attack it. For example, they frequently deliver the lung out of the incision; they also pack in gauze compresses. They lose sight of the fact that the size of the incision and the size of the opening are not synonymous when anything is in the incision, no matter whether it is a delivered lung, a gauze compress, the surgeon's hand, instruments or what not. A hole that is either partly or completely plugged obviously cannot be as large as if it contained nothing. Other aids in combating the asphyxial effects of an open pneumothorax are the administration of either oxygen or carbon dioxide, as recommended by Dautrebande and Spehl, to break up the vicious circle which occurs when the anoxemia appears. Oxygen will supply directly the thing that is most needed, but the administration of carbon dioxide will accomplish the same purpose indirectly by stimulating deeper inspirations and thereby the taking in of more air.

In this general summary of the effects of alterations of intrapleural pressure which has been given above it is recognized that some of the ideas and theories here set forth may perhaps have to be modified from time to time as later work sheds new light on the subject. At the present time, however, it seems to me that no general theory of the action of pneumothorax explains all the known facts so well

as the one which has been advanced above. In principle the theory is the same as that expressed in 1918; it differs only in relatively unimportant details. Those who have disagreed with the principles of the theory have invariably failed to offer a constructive alternative hypothesis which is at all in accord with all the known facts concerning the action of pneumothorax.

BIBLIOGRAPHY

[1]Lilienthal and Ware, Recent Progress in the Operative Treatment of Empyema of the Thorax, Med. Rec., 1916, vol. 90, p. 89.

[2]Wilensky, Empyema of the Thorax, Surg. Gyn., and Obst., 1915, vol. 20, p. 501.

[3]Empyema Commission of U. S. Army, Cases of Empyema at Camp Lee, Va., Jour. A. M. A., 1918, vol. 71, pp. 366 and 443.

[4]MacCallum, W. G., The Pathology of the Pneumonia in the United States Army Camps During the Winter of 1917-18, Monographs of the Rockefeller Institute for Medical Research, 1919, No. 10.

[5]Graham, E. A., and Bell, R. D., Open Pneumothorax: Its Relation to the Treatment of Empyema, Am. Jour. Med. Sci., 1918, vol. 156, p. 839.

[6]Donders, quoted by Heynsius, Arch. f. d. ges. Physiol., 1882, vol. 29, p. 267.

[7]Aron, Die Mechanik and Therapie des Pneumothorax, 1902, quoted by Emerson[8], p. 194.

[8]Emerson, Pneumothorax, Johns Hopkins Hosp. Rep., 1903, vol. 11, p. 1.

[9]Sauerbruch, Zur Pathologie des offenen Pneumothorax und die Grundlagen meines Verfahrens zu seiner Ausschaltung, Mitteil, a. d. Greuz geb. d. Med. u. Chir., 1904, vol. 13, p. 399.

[10]Mayer, L., Les Bases Physiologiques de la chirurgie Pleuropulmonaire, Ann. d. l. Soc. Roy. d. Sciences Med. et Nat. de Bruxelles, 1906, vol. 15, p. 1.

[11]Garré, Der Pneumothorax, III Congres de la Societé internationale de chirurgie, 1911, p. 1; also in "Surgery of the Lungs."

[12]See 10.

[13]Elsberg, Pneumothorax and Posture, Jour. Exper. Med., 1909, vol. 11, p. 444.

[14]See 8.

[15]Howell, Text-book of Physiology, Philadelphia, 1911, p. 646.

[16]Peabody and Wentworth, Clinical Studies of the Respiration, IV. The Vital Capacity of the Lungs and Its Relation to Dyspnea, Arch. Int. Med., 1917, vol. 20, p. 443.

[17]See 9.

[18]Sackur, Weiteres z. Lehre von Pneumothorax, Arch. f. path. Anat. u. Physiol., 1897, vol. 150, p. 151.

[19]Stone, Pericarditis as a Complication in Pneumonia, Jour. A. M. A., 1919, vol. 73, p. 254.

[20]See 3.

[21]Stone, Management of Postpneumonic Empyema Based upon 310 Cases, Am. Jour. Med. Sci., 1919, vol. 158, p. 1.

[22]Diederich, Surg., Gyn. and Obst., 1919, vol. 28, p. 362.

[23]Stevens, F. A., Recurrences after Operations for Empyema, Jour. A. M. A., 1919, vol. 73, p. 813.

[24]Stewart, G. A., Treatment of Empyema by Carrel-Dakin Method, Med. Rec., 1918, vol. 94, p. 236.

[25]Tuffier, Treatment of Chronic Empyema, Trans. Am. Surg. Assn., 1919.

[26]Moschcowitz, A. V., The Surgical Treatment of Empyema, Surg. Gyn. and Obst., 1919, vol. 28, p. 337.

[27]Heuer, G., Observations on the Treatment of Chronic Empyema, Trans. Am. Surg. Assn., 1919.

[28]Woodyatt, R. T., Personal communication.

[29]Opie and Alford, Influence of Diet on the Toxicity of Substances which Produce Lesions of the Liver or the Kidney, Jour. A. M. A., 1914, vol. 63, p. 136.

[30]Graham, E. A., The Resistance of Pups to Late Chloroform Poisoning in Its Relation to Liver Glycogen, Jour. Exper. Med., 1915, vol. 21, p. 185.

[31]Whipple, G. H., Insusceptibility of Pups to Chloroform Poisoning During the First Three Weeks of Life, Jour. Exper. Med., 1912, vol. 15, p. 259.

[32]Graham, E. A., and H. T., Retardation by Sugars of Diffusion of Acids in Gels, Jour. Amer. Chem. Soc., 1918, vol. 40, p. 1900.

[33]See in this connection the article by Woodyatt, Jour. A. M. A., 1910, vol. 55, p. 2109.

[34]Balcar, Sansum and Woodyatt, Fever and the Water Reserve of the Body, Arch. Int. Med., 1919, vol. 24, p. 116

[35]Moynihan, Sir Berkely, Gunshot Wounds of Lungs and Pleura, Surg., Gynec. and Obst., 1917, vol. 25, p. 605.
[36]Duval, P., Abstr. in Review of War Surgery and Medicine, Office of the Surgeon General, Washington, 1918, vol. 1, No. 4, pp. 1 to 27.
[37]Piéry, Quoted from Review of War Surgery and Medicine, loc. cit., p. 10.
[38]Ibid., p. 14.
[39]Roberts and Craig, ibid., p. 18.
[40]Anderson, ibid., p. 20.
[41]Gask and Wilkinson, ibid., p. 22.
[42]Stivelman and Rosenblatt, Protrusion of Artificial Pneumothorax into the Opposite Untreated Side, Jour. A. M. A., 1919, vol. 72, p. 1445.
[43]Matas, R., The Value of Artificial Aids to Respiration in Acute Operative Collapse of the Lungs, Arch. Surg., 1922, vol. 5, p. 110.
[44]Duval, P., Les données actuelles de la chirurgie intrathoracique unilaterale en plèvre libre, Presse Méd., 1922, vol. 30, p. 409.
[45]Yates, J. L., Effects of Acute and Chronic Pneumothorax. A Preliminary Report, Am. Jour. Med. Sci., 1923, vol. 165, p. 1.
[46]Snyder, J. W., Studies in Intrapleural Tension, Arch. Surg., 1924, vol. 8, p. 364.
[47]Duval, P., Les plèvres, communiquent-elles normalement chez le chien? Presse Méd., 1923, No. 68, p. 733.
[48]Garland, G. M., Pneumodynamics, Hurd and Houghton, New York, 1878, p. 53.
[49]Calvert, J., On the Intrapleural Pressure and Manner of Contraction of the Lung in Pleural Effusion. St. Barth. Hosp. Rep., 1892, vol. 28, p. 131.
[50]Graham, E. A., A Reconsideration of the Question of the Effects of an Open Pneumothorax. Arch. Surg., 1924, vol. 8, p. 345.
[51]Graham, E. A., Principes qui découlent de la chirurgie intrathoracique. Presse Méd., 1923, vol. 31, p. 141.
[52]Haldane, J. S., and Poulton, E. P., The Effects of Want of Oxygen on Respiration. J. Physiol., 1908, vol. 37, p. 390.

[53]Haldane, J. S., Meakins, J. C., and Priestley, J. G., The Effect of Shallow Breathing, J. Physiol., 1919, vol. 52, p. 433.

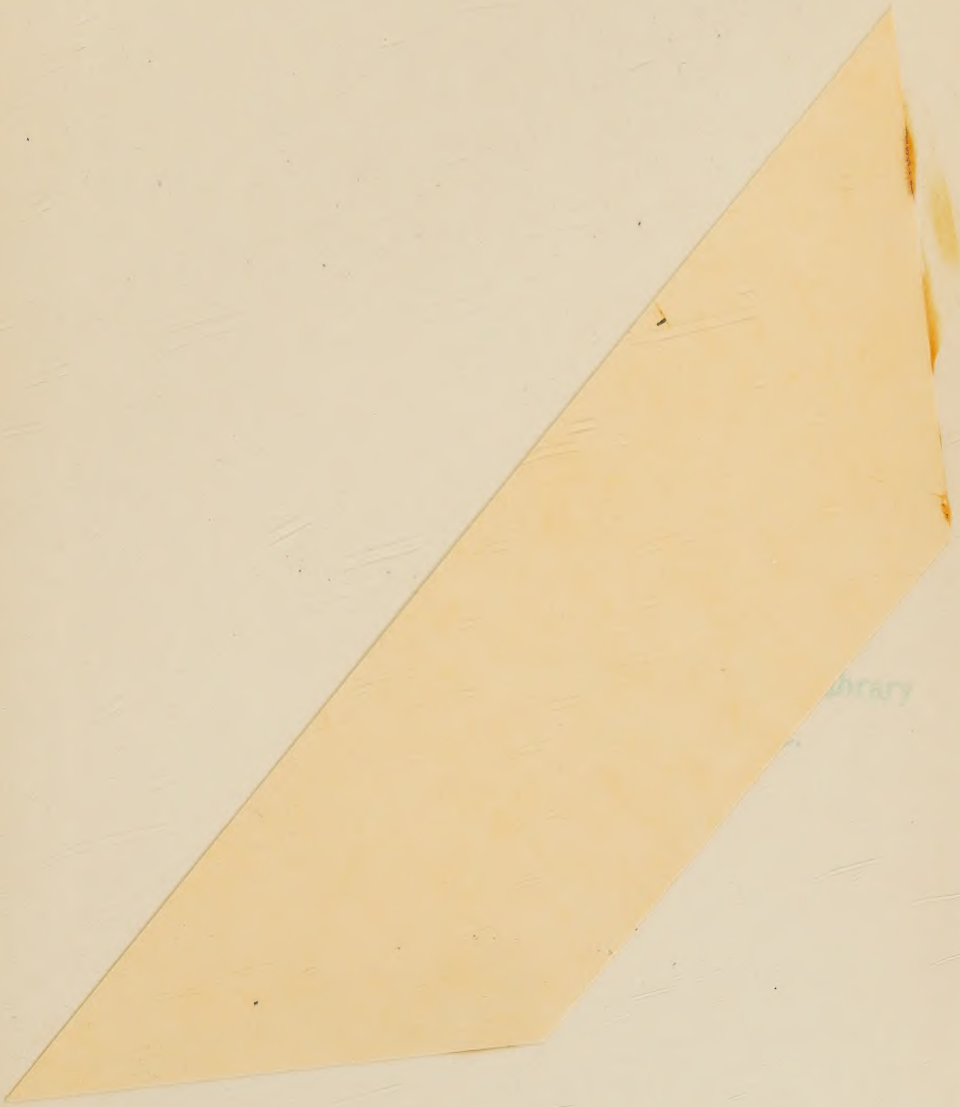
[54]Dautrebande, L., and Spehl, P., Les causes de la dyspnée et de la mort dans le pneumothorax ouvert. Bull. Acad. roy. de méd. de Belg., 1922, 5, vol. 10, pt. 2, p. 533.

[55]Lundsgaard, C., and Van Slyke, D.D., Cyanosis, Medicine, 1923, vol. 2, p. 1.

[56]Sackur, Zur Lehre vom Pneumothorax. Ztschr. f. klin. Med., 1896, vol. 29, p. 25.

MEDICAL LIBRARY OF
MECKLENBURG COUNTY, INC.

2313



MEDICAL LIBRARY OF
MECKLENBURG COUNTY, INC.

C0-ARK-336